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CLINICAL THERMOMETERS AND CROSS-INFECTION IN HOSPITALS*

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Cross-infection in hospitals, particularly in childrens' wards is always a serious hazard. The main sources of infection are patients, attendants and visitors.[1] Infection may also be spread by non-human agencies such as thermometers, and instruments commonly used in examinations such as spatulas, stethoscopes and auriscopes.

In studying the clinical thermometer as a possible source of cross-infection, several workers have investigated the antiseptic fluid in which the thermometer is kept. Rubenstein and Foley[6] found that bacterial counts made on one of the thermometer fluids (1 in 10,000 bichloride of mercury solution) revealed a bacterial population of 7 million viable organisms per c.c., the majority of which were *Escherichia coli*. Similarly, Green and Penfold[3] found thermometer fluids (chiefly Glyc. thymol. co.) grossly contaminated with various pathogenic and non-pathogenic organisms. Both investigations were carried out in wards in which 'communal' thermometers were used, i.e. wards in which a few thermometers were shared by a group of patients.

In the present investigations, attention was paid to the *thermometer itself* as a possible cause of infection, since culture of the thermometer ends which go into the patient's mouth or rectum should give a better estimate of bacterial contamination than culture of the antiseptic fluids.

PRESENT INVESTIGATION

The work was carried out in a general hospital. In the paediatric wards each child has his own thermometer, but 'communal' thermometers are still in use in other wards. The antiseptic used is 1 in 1,000 biniodide of mercury, supplied by the dispensary. There is considerable variation in the frequency with which the antiseptic is changed; it is usually 2 or 3 times a week.

A number of serum-culture tubes were supplied by the bacteriology department. Visits were made to the wards without any notice being given, usually before the 10 a.m. temperature round, so that the thermometer had been immersed in the antiseptic for approximately 4 hours. In the examination of a thermometer, the bung of a tube was removed and the thermometer taken out of its jar and dipped for about half its length into the tube. The bung was at once replaced and the thermometer put back into its jar. The serum tubes were then taken to the bacteriology department for culture.

The total number of thermometers tested was 109, of which 63 were found to be contaminated. The results obtained from examination of oral and rectal thermometers were as follows:

Oral		*Rectal*	
Number investigated	51	Number investigated	58
Number contaminated	28	Number contaminated	35

The organisms grown from oral and rectal thermometers were as follows:

	Rectal	*Oral*	*Total*
Coagulase-positive *M. pyogenes aureus**	7	—	7
Coagulase-negative *M. albus*	8	4	12
Enterococci	15	—	15
Viridans streptococci	—	15	15
Haemolytic streptococci	1	—	1
Diphtheroids	—	7	7
Pseudomonas aeruginosa	1	—	1
Escherichia coli	1	—	1
Aerococcus	—	1	1
B. rotans	—	1	1
Sarcinae	2	—	2
	35	28	63

Mixed infections occurred in 12 instances.

* A paper presented at the South African Medical Congress, Pretoria, October 1955.

* 'Hospital staph.', sentitive to chloramphenicol and erythomycin, but not to penicillin, streptomycin, oxytetracycline or sulphatriad.

These results seem to show that even after several hours' immersion in antiseptic (1 in 1,000 biniodide solution) over half the thermometers investigated were contaminated with pathogenic and non-pathogenic organisms. This applies to both oral and rectal thermometers.

It is likely the contamination would be even higher if tests were taken during a temperature round since, under the 'communal' thermometer system, very little time is allowed for the thermometer to remain in the fluid before use.

DISCUSSION

In order to reduce the possibility of cross-infection from contaminated clinical thermometers, two steps would have to be taken. In the first place, the 'communal' thermometer would have to be abolished and replaced by a system in which each patient would have his own thermometer. Secondly, the fluid used for sterilizing the thermometer would have to be effectively germicidal and suitable for clinical use. Biniodide of mercury is still largely used for this purpose, but mercurial compounds fall far short of being ideal germicides, though the belief persists that they are highly effective germicides.[2]

It is not proposed to discuss the question of a suitable antiseptic in detail, except to mention that tests are being made with 2 cationic detergents, viz. Cetrimide (Cetavlon), and Benzalkonium chloride (Zephiran). They reduce surface tension and have bactericidal properties. In the dilutions normally used, both are non-irritating and non-toxic to raw surfaces.[1] Preliminary observations have shown a marked reduction in the number of contaminated thermometers where these detergents were used as thermometer fluids.

In ideal circumstances, each patient should have his own thermometer, but there are certain objections to the keeping of a thermometer at the patient's bedside. Nurses of experience maintain that breakages would be frequent, patients or visitors might tamper with the fluid, and nervous patients might be constantly taking their own temperatures. In a large ward it is not easy to keep thermometers free from dust, and after a patient has been discharged, the nurse may easily forget to change the fluid for the next patient. Some of these objections apply even more with children, particularly older children. It is very difficult to keep anything out of the reach of an active convalescing child.

In order to overcome certain of the difficulties mentioned, the author has suggested the use of a portable stand, based on the principle that each patient has his own thermometer.[5] All the thermometers are immersed in tubes on the stand, which can be kept in a cupboard out of reach of the patient. It is easy to keep everything clean, and changing the solution is an easy process which can be done quickly and efficiently at one time. To simplify the taking of termperatures in large wards, each tube and its thermometer are clearly numbered, corresponding with the bed or cot. The nurse in charge can thus be readily guided while she is taking temperatures in a ward; e.g., the patient in bed No. 1 receives thermometer No. 1 which, after use, goes into tube No. 1.

CONCLUSIONS AND SUMMARY

1. Cultures of oral and rectal thermometers in a general hospital showed that more than half were contaminated with pathogenic and non-pathogenic organisms. The thermometer fluid currently in use is 1 in 1,000 biniodide of mercury, and 'communal' thermometers are still used in some wards.

2. Suggestions are made regarding the prevention of cross-infection by clinical thermometers. Each patient should have his own thermometer and beds should be numbered to prevent confusion. The use of a portable stand for ward use is described.

The work was done with the kind permission of Dr. N. H. G. Cloete, and Professor F. Ford. Thanks are due to Dr. A. Kipps for the bacteriological reports and for helpful advice.

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POLIOMYELITIS VACCINE, WINTER 1956

A letter from Dr. J. H. S. Gear, Director of Research of the Poliomyelitis Research Foundation Laboratories, P.O. Box 1038 Johannesburg, under date 23 April states:

'The issue of poliomyelitis vaccine from the Laboratories of the Poliomyelitis Research Foundation will be resumed as from 1 July 1956. This vaccine will be issued to local authorities and to medical practitioners.

'The vaccine is of a formalin-inactivated type, prepared from suspensions of poliovirus grown in tissue cultures of monkey kidney cells. This vaccine conforms with the minimum requirements laid down by the Union Health Department for poliomyelitis vaccine. These are based on the latest United States Public Health Service minimum requirements for poliomyelitis vaccine.

'A course of 3 inoculations of 1 c.c. each is recommended, with an interval of 1-2 months between the 1st and 2nd, and 6 months or more between the 2nd and 3rd inoculations. A certain amount of protection is conferred by one injection, still more by 2, but to get the maximum effect it is desirable to give 3 inoculations.

'The procedure to be followed by medical practitioners in applying for supplies of vaccine is as follows: A list of the children for whom vaccine is required should be sent to the Poliomyelitis Research Foundation, P.O. Box 1038, Johannesburg, giving their names, ages, and parents' occupations.

'If the demand does not exceed the supply there will be no limitation on the issue of the vaccine, but if the demand does exceed the supply it will be issued according to the following priorities:

'*First Priority:* Children up to the age of 6, and the children of doctors, nurses, teachers, and health officials, up to the age of 16.

'*Second Priority:* Children in the age-group 6-10 years.

'*Third Priority:* Children in the age-group 11-15 years.

All local authorities will be sent a circular asking for details of their needs.'

South African Medical Journal

Suid-Afrikaanse Tydskrif vir Geneeskunde

EDITORIAL

INTERSEX

In an article in this issue (page 417) Drs. R. Hoffenberg and W. P. U. Jackson consider certain aspects of our modern knowledge of intersex or hermaphroditism. Although some authorities would disagree, on the whole these two terms are interchangeable. The more romantic Greek word is taken from the mythological intersexual offspring of Hermes, the precocious messenger of the gods, and Aphrodite, the goddess of love.

Romantic, however, these patients are certainly not, and it is our duty to do as much for them as we can by making a definite diagnosis as early as possible, and then by monosexualizing with hormones and plastic surgery at the appropriate time. Money, Hampson and Hampson[2] have recently and painstakingly shown that the most important aspect in the management of the case is its early assignment to the male or female sex and its rearing in the assigned sex. They claim that by the age of 2 years the child is already becoming imbued with the attributes of the sex in which it is being reared. Further, whatever the type of intersexuality, it is very unusual for an individual later to desire to be of the sex opposite to that in which he or she has been reared. Thus Money *et al.*[3] found only 4 cases out of 76 whose psychological desire went against their assigned sex. Hoffenberg and Jackson's patient 'L.R.' was plainly unusual in this respect.

The whole subject has become far more interesting owing to several recent advances in our knowledge. First there have been more accurate analyses of the purely morphological aspects of the different types of intersex, and the interested reader is especially referred to two articles in the same journal, one from Johns Hopkins Hospital, Baltimore (Wilkins *et al.*[4]) and the other from the Boston Childrens' Hospital (Gross and Meeker[1]). It is plain that the old classification of intersex into true hermaphroditism (with both ovarian and testicular tissue), male pseudo-hermaphroditism (with testicular tissue only), and female pseudo-hermaphroditism (with ovarian tissue only) is not sufficient for completeness, but is still a good working basis. Both Wilkins and Gross prefer to classify quite separately

VAN DIE REDAKSIE

INTERSEKSUALITEIT

Sekere aspekte van ons hedendaagse kennis van interseksualiteit of dubbelslagtigheid word in 'n artikel in hierdie uitgawe (bls. 417) deur drs. R. Hoffenberg en W. P. U. Jackson bespreek. Hoewel sommige gesaghebbendes nie sal saamstem nie, is hierdie twee benamings oor die algemeen wisselbaar. Die meer romantiese Griekse woord is afkomstig van die mitologiese dubbelslagtige nakomelinge van Hermes, die vroegrype boodskapper van die gode, en Aphrodite, die godin van die liefde.

Romanties is hierdie pasiënte egter nie. Dit is ons plig om ons bes te doen om hulle te help deur die afwyking so vroeg moontlik te diagnoseer, en om hulle dan met hormone en plastiese chirurgie te geleëner tyd eenslagtig te maak. Onlangs het Money, Hampson en Hampson[2] noukeurig bewys dat vroegtydige indeling as óf manlik óf vroulik, en opvoeding volgens die beoordeelde geslag, die belangrikste aspek van die behandeling van so 'n geval is. Hulle meen dat 'n kind reeds op tweejarige ouderdom die kenmerke van die geslag waarvolgens hy grootgemaak word, begin aanneem. Afgesien van die soort interseksualiteit, is dit ook baie seldsaam dat 'n indiwidu wat in die een geslag opgevoed is, later begeer om aan die teenoorgestelde geslag te behoort. Money en sy medewerkers[3] het dan ook slegs 4 uit 76 gevalle teëgekom wie se sielkundige begeerte nie met hul toegeskrewe geslag ooreengekom het nie. Dit is duidelik dat Hoffenberg en Jackson se pasiënt ‚L.K.' in hierdie opsig buitengewoon was.

Die onlangse vooruitgang in ons kennis het hierdie onderwerp baie interessanter gemaak. Eerstens was daar die juister ontledings van die suiwer morfologiese aspekte van die verskillende soorte dubbelslagtigheid, en die belangstellende leser word veral verwys na twee verhandelinge wat in dieselfde tydskrif verskyn, die een uit die Johns Hopkins-hospitaal, Baltimore (Wilkins *et al.*[4]), en die ander een uit die Bostonse Kinderhospitaal (Gross en Meeker[1]). Dit is duidelik dat die eertydse indeling van interseksualiteit in egte dubbelslagtigheid (met beide eierstok- en testes-weefsel), manlike skyndubbelslagtigheid (alleenlik met testesweefsel), en vroulike skyndubbelslagtigheid (met slegs eierstokweefsel) nie afdoende is nie, maar dit is nog altyd 'n gangbare grondslag. Beide Wilkins en Gross verkies om vroulike gevalle van skyndubbelslagtigheid, wat aan die bynier-geslagssindroom te wyte is (verreweg die meerderheid), heeltemal apart te klassifiseer as

those cases of female pseudo-hermaphroditism which are due to the adreno-genital syndrome (and these are the great majority) as examples of intersexual development caused by hormonal influences. However, there are apparently two other, much rarer, types of hermaphroditism associated with abnormal hormonal production—female pseudo-hermaphrodites born to mothers with androgen-secreting arrhenoblastomas and male pseudo-hermaphrodites whose testes secrete primarily oestrogens. Other varieties of intersex which do not quite fit into this simple classification include total agonadism[1] and the otherwise normal male who shows complete absence of the penis.[1] An error in diagnosis in the latter case should be avoided by the finding of testes, which are usually descended. Finally, we now believe that the condition of gonadal dysgenesis ('ovarian agenesis') represents the extreme degree of male pseudo-hermaphroditism, as discussed in this *Journal* in a recent editorial.[5]

A second recent advance has concerned the female pseudo-hermaphrodites, in almost all of whom bilateral adrenal hyperplasia occurs in intra-uterine life. Bongiovanni[6] has shown that there is a biochemical block in the formation of hydrocortisone in these adrenals, so that a steroid precursor, hydroxyprogesterone, or a related compound, cannot be further metabolized, and so exists in excessive quantity. These precursors of hydrocortisone are androgenic, thus accounting for the masculinization of the foetus, with persistence and development of Wolffian-duct structures. At the same time, the usual pituitary inhibitor or 'regulator', hydrocortisone, is largely missing, so that ACTH is produced in excess, and this stimulates a general hyperplasia of the adrenal cortex. Wilkins was the first to utilize the clinical application of this theory, and he has shown that continued cortisone therapy will produce an almost complete conversion of these male-looking hermaphrodites to true females,[7] even to the extent of normal pregnancy. Only one feature remains for surgical correction—that is an over-large phallus. This condition is unlike other varieties of intersex in being quite frequently familial—probably on the basis of an inherited recessive gene. Hoffenberg and Jackson remark upon the great diagnostic importance of a high urinary 17-ketosteroid output in this state.

The third advance is in connection with the ability to diagnose the genetic sex of an individual by the microscopical examination of skin, or even a vaginal scraping or blood smear. The great value of this is at once plain, and has recently been discussed in our columns.[8] It has proved very helpful in the diagnosis of the 3 main varieties of hermaphrodite. It does not mean, however, that a person with, say, a male skin-sex must necessarily be considered as a male person

voorbeelde van tussengeslagtelike ontwikkeling veroorsaak deur die invloed van hormone. Daar is egter klaarblyklik nog twee ander soorte dubbelslagtigheid wat baie seldsamer is en wat in verband staan met abnormale hormoonproduksie—vroulike skyndubbelslagtiges gebore uit moeders met androgeen-afskeidende geswelle in die geslagskliere, en manlike skyndubbelslagtiges wie se testes hoofsaaklik estrogene afskei. Ander soorte dubbelslagtigheid wat nie geredelik by hierdie eenvoudige klassifikasie ingepas kan word nie, sluit in algehele afwesigheid van die geslagskliere,[1] en die andersins normale man by wie die penis heeltemal ontbreek. By laasgenoemde geval kan 'n verkeerde diagnose vermy word deur die vind van testes, wat gewoonlik afgedaal is. Ten laaste meen ons vandag dat die toestand van gebrekkige ontwikkeling van die geslagskliere (‚onontwikkeling van die eierstokke') die uiterste graad van manlike skyndubbelslagtigheid is. Dit is onlangs in die Redaksie-rubriek in hierdie *Tydskrif* bespreek.[5]

'n Tweede vooruitgang is onlangs gemaak aangaande vroulike skyndubbelslagtigheid, waarby daar in nagenoeg alle gevalle 'n tweesydige, oormatige groei van die byniere gedurende die voorgeboortelike periode plaasgevind het. Bongiovanni[6] het aangetoon dat daar 'n biochemiese blokkade in die vorming van hidrokortisoon in hierdie byniere is, sodat 'n steroïed-voorloper, hidroprogesteroon, of 'n verwante verbinding nie deur die stofwisseling verder verwerk word nie, met die gevolg dat daar 'n oormaat daarvan bestaan. Hierdie voorlopers van hidrokortisoon is androgene, en is dus verantwoordelik vir die vermanliking van die vrug, met die gevolglike voortbestaan en ontwikkeling van Wolff se buis. Terselfdertyd is hidrokortisoon, die gewone hipofise-stremmer of ‚reguleerder', grotendeels afwesig, sodat 'n oormaat ACTH opgebou word, en dit is weer verantwoordelik vir 'n algemene oorontwikkeling van die bynierskors. Wilkins was die eerste om gebruik te maak van die kliniese toepassing van hierdie teorie, en hy het bewys dat aanhoudende behandeling met kortisoon hierdie mansagtige dubbelslagtiges amper geheel en al in ware vroulikes kan laat verander,[7] in so 'n mate dat hulle selfs normaal swanger kan word. Dan is slegs 'n enkele chirurgiese verbetering nodig—die korrigeer van 'n oorgroot manlike lid. Hierdie kondisie verskil van die ander soorte interseksualiteit in die opsig dat dit taamlik dikwels 'n familietrek is—waarskynlik op die grondslag van 'n oorgeërfde resessiewe geen. Hoffenberg en Jackson beklemtoon die belangrikheid by diagnose van 'n hoë urine-uitskeiding van 17-ketosteroïede by hierdie toestand.

Die derde vooruitgang staan in verband met die vermoë om 'n persoon se genetiese geslag te diagnoseer deur middel van 'n mikroskopiese vel-ondersoek, of selfs deur 'n skede-skraapsel of bloedsmeer. Die groot waarde hiervan is sonder meer duidelik, en is onlangs in ons *Tydskrif* bespreek.[8] Dit het baie waardevol geblyk by die uitkenning van die drie hoofsoorte van dubbelslagtigheid. Dit beteken egter nie dat 'n persoon wat byvoorbeeld geneties 'n manlike vel-geslag het as manlik beskou en behandel moet word nie. Dit sou

and so treated. This would lead to the absurd conclusion of transforming a patient with gonadal dysgenesis into a man, when she is female by inclination, upbringing, external and internal accessory sexual organs, and general body-shape. In fact, a consideration of the problem makes us realize that no particular attribute makes a male or a female. Features which we would usually consider fundamental in one sex may actually be at variance with each other. Thus we have the genetic sex, the gonadal sex, the hormonal sex, the sexual pattern of the internal genitalia, the sexual pattern of the external genitalia, the sex in which the person is reared and, finally, the patient's own chosen sex. All these must be considered together before deciding which is the 'best' sex to be adopted in each case (not the 'correct' sex, since this term is meaningless). Sex is no longer as simple as it seems!

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5. Editorial (1956): S. Afr. Med. J., **30**, 285.
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7. Wilkins, L., Gardner, L. I., Krigler, J. S. Jnr., Silverman, S. H. and Migeon, C. J. (1952): *Ibid.*, **12**, 257 and 277.
8. Editorial (1955): S. Afr. Med. J., **29**, 809.

neerkom op die onsinnigheid dat 'n pasiënt met gebrekkige ontwikkeling van die geslagskliere vermanlik word, hoewel sy volgens begeerte, opvoeding, inwendige en uitwendige bykomende geslagsorgane, en algemene liggaamsbou, vroulik is. Nadenke oor hierdie probleem dwing ons juis tot die gevolgtrekking dat 'n mens nie sy manlikheid of haar vroulikheid aan één, afsonderlike hoedanigheid te danke het nie. Die trekke wat ons gewoonlik as fundamenteel by die een geslag beskou, kan in werklikheid onderling teenstrydig wees. Daar is byvoorbeeld die genetiese geslag, die kliergeslag, die hormoongeslag, die seksuele patroon van die inwendige geslagsorgane, die seksuele patroon van die uitwendige geslagsdele, die geslag waarvolgens die persoon opgevoed is, en ten laaste die geslag wat die pasiënt self verkies. Al hierdie feite moet tesame in ag geneem word voordat daar besluit word op die ‚beste' geslag vir elke geval (nie die ‚ware' geslag nie, want die term het geen betekenis nie). Geslag is nie meer so eenvoudig as wat dit voorkom nie!

1. Gross, R. E. en Meeker, I. A. (1955): Pediatrics, **16**, 303.
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THE PROBLEM OF INTERSEX

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Intersex is not uncommon. Young [24] stated that about one child in every thousand exhibited ambisexual genital configuration. The vast majority of these are pseudo-hermaphrodites. In recent years many advances have been made in the understanding of the problem of intersex, with improvements in diagnostic techniques and in methods of management. In every case the exact reasons for assignment to one or other sex can now be accurately assessed and, indeed, must be so assessed. Vacillation of parents or doctor regarding the sex in which a child should be brought up, with consequent shunting of the patient from male to female and back again, can produce only misery.

Even in those cases in which a definite sex assignment is made—and adhered to—changes at puberty may occur in an unexpected direction. Thus, a 'schoolgirl' may betray her maleness only at puberty, with the growth of facial hair, deepening of the voice and masculine bodily development. If these tragedies are to be avoided, an understanding of the nature of such intersexual states is necessary. It is the purpose of this paper to recount the histories of two patients with pseudo-hermaphroditism, to present some of the current views on intersex, and to describe some of the methods which permit of more accurate diagnosis.

Types of Intersex

Three main categories exist:

1. *True Hermaphrodites.* Gonads of both sexes are present; these may be an ovary on one side with a contralateral testis; or one or both of the gonads may be an ovotestis (i.e. a gonad containing both ovarian and testicular elements).

2. *Female Pseudo-hermaphrodites.* Here both gonads are ovaries, but there is development of some male accessory organs (Wolffian duct derivatives).

3. *Male Pseudo-hermaphrodites.* Both gonads are testes, but female genital ducts and/or external genitalia are found.

TRUE HERMAPHRODITES

These constitute the rarest types of intersex, only about 60 authentic cases having been reported in the literature. Bilateral gonads are present, which may be intra- or extra-abdominal; very rarely a third gonad has been

found. The phallus is variable in size and form; always there is penoscrotal hypospadias. The majority show a bifid scrotum; in some, however, the labio-scrotal folds surround a short phallus and resemble normal labia around a larger than normal clitoris. Almost invariably patients have a normally formed, although small, uterus with one or two fallopian tubes; rare exceptions have been described.[17] Puberty may ensue in either direction. Exploratory laparotomy with bilateral gonadal biopsy is generally needed to confirm the diagnosis.

FEMALE PSEUDO-HERMAPHRODITES

Two forms of this condition are described:

1. The vast majority are the result of intra-uterine adrenocortical overactivity. As a result of this, excessive androgen outpouring leads to more or less evidence of masculinization. This excessive hormonal influence, if not treated, persists throughout life. The external genitals are almost normally female, except for enlargement of the clitoris; in gross instances this may be mistaken for a penis with hypospadiac scrotal development. Rarely a penile urethra is found with a fused, empty, scrotal sac; this variant is usually misdiagnosed as cryptorchidism.

These patients generally have a precocious puberty of male type. More than one member of a family may be affected. Because of the excessive androgen production the urinary 17-ketosteroids are invariably high. This permits certain diagnosis to be made in early post-natal life.

The basic metabolic fault in this condition has been elucidated recently.[4] A block in the synthesis of hydrocortisone has been shown to exist, so that an androgenic precursor is released into the circulation. This substance (hydroxyprogesterone or a related compound) is responsible for the virilization.

Because of the lack of hydrocortisone, episodes of acute adrenal insufficiency of an Addisonian type (with vomiting, diarrhoea and collapse) may occur in the infant. Attacks of this sort in a child of ambiguous sexual development make the diagnosis of adrenogenital syndrome obligatory.

2. Very rarely adrenal overactivity cannot be demonstrated in cases of female pseudo-hermaphroditism.[13] Puberty may then occur in a female direction and reproduction may be possible. In these patients 17-ketosteroid excretion is normal. This form, however, is exceedingly rare and is difficult to distinguish from true hermaphroditism without exploratory laparotomy and gonadal biopsy. No adequate explanation of this type of female pseudo-hermaphroditism has been proposed, although it has been postulated that the female foetus undergoes partial intra-uterine masculinization because of a temporary phase of adrenal androgenic overactivity. At birth this is no longer detectable and the 17-ketosteroid excretion is thus normal. This theory is an ingenious one, but there is no real evidence to support it.

These two forms must be diagnosed early in life, as the adrenocortical type is totally correctable with cortisone therapy and, in both types, reproduction is possible.

MALE PSEUDO-HERMAPHRODITES

This condition exists in several different degrees. It has been shown[11,12] in experimental animals that removal or ablation of the gonads at an early stage of embryonic life (i.e. before differentiation of the genital tract is complete) is followed by the development of the female body-form. If this 'castration' occurs very early, complete feminization ensues, whether the embryo was destined genetically to be male or female. If it occurs slightly later, partial masculine differentiation having already taken place, greater or lesser degrees of intersexual development will be seen. In other words, the earlier the 'castration' occurs, the less obvious are the male features and, in extreme cases, slight enlargement of the clitoris may be the only token of the basic maleness of the animal.

It is suggested that comparable happenings in the human male embryos (i.e. intra-uterine damage to the developing gonads at different stages in different cases) may produce varying degrees of feminization. The genetic maleness of the subject may be determined by use of the skin 'sexing' method (*vide infra*). Pubertal development occurs along normal male lines, but may be partially eunuchoidal.

Another very rare form of male pseudo-hermaphroditism is found where the testes actually produce oestrogens.[13] These patients may have completely normal female genital appearance and they comprise the only members of this group who have a feminizing puberty. Since they live as normal women, this course should not be interrupted. The testes in these individuals may remain intra-abdominal or may present in the groin. Occasionally these patients show the external genital appearance of cryptorchid, hypospadiac males and, in such cases, pubertal feminization reveals their true status.

CASE REPORTS

Case 1

M.E. European 27 years old. This patient, despite original uncertainty, was raised as a girl. When she was 14 years old, a practitioner considered her to be female, amputated a fairly large phallus and removed a gonad which was present in the left labio-scrotal fold; a similar gonad in the right fold was left alone. A year later puberty commenced—along masculine lines. Neither breasts nor menses appeared; instead the voice deepened, facial hair began to grow and soon advanced to a degree which required daily shaving; the body contours assumed a muscular male configuration. Oestrogen therapy was instituted and, in the following few years, some breast development ensued. There was, however, no diminution in the degree of masculinity.

The patient continued to live and work as a female when she left school. She herself entertained doubts about her true sex and, despite being physically attracted towards men, actually attempted mild and 'experimental' sexual relationships with both men and women. About 2 years ago she met a man whom she wishes to marry. Moderate physical intimacy has taken place and it is this friendship which precipitated her admission to Groote Schuur Hospital in the hope that more complete feminization could be established.

On examination, she presented as an attractive but very hirsute female (Fig. 1); axillary and pubic hair was profuse—the latter

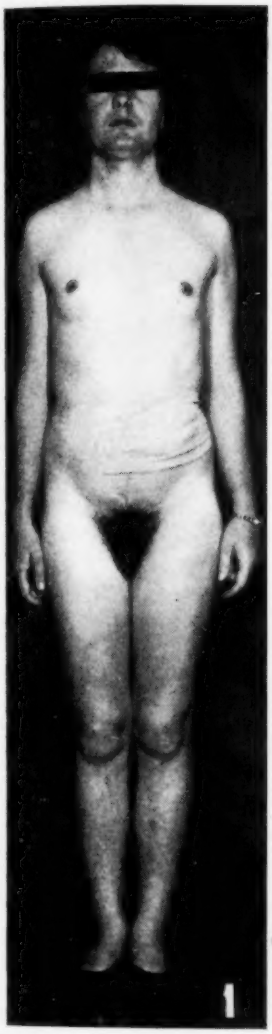

Fig. 1. Case 1 (M.E.).

of typically male distribution. The limbs were muscular and hairy, the breasts poorly developed with dark areolae. External genitalia (Figs. 2 and 3) comprised hypertrophied labio-scrotal folds enclosing a rudimentary vagina and, anteriorly, the scar of the amputated phallus. In the right fold, a bean-sized gonad was felt with an apparent epididymis attached. Further examination showed a small, blind vaginal pouch, but no other evidence of Mullerian derivatives.

17-Ketosteroid estimation was normal and skin biopsy showed a male type of chromatin pattern.

A psychological appraisal was undertaken (Dr. S. Berman, Chief of the Department of Neuropsychiatry). The conclusions reached were that the patient displayed femininity of outlook and it was felt that one should assist her in her desires to become 'normally' female.

At operation, Mr. N. Peterson (Chief of the Plastic Surgery Unit) performed vaginoplasty and excised the remaining gonad. Histology revealed this to be a degenerate testis (a finding probably related to the prolonged oestrogen therapy).

Comment. It is plain that this patient was a male with extreme hypospadias, a bifid scrotum, and testes which had descended into the scrotal folds. (Such normal descent is most uncommon in this sort of case.) This appearance led to the misdiagnosis of her sex with consequent mutilation which forced upon her the choice of continuing life as a male without a penis or a bearded, infertile female. The doctor had removed what he thought to be an enlarged clitoris and also a scrotal testis. This, combined with her strong predilection for femaleness, left us with no option but to assist her surgically and hormonally to attain this goal. Maintenance therapy with oestrogens will, perhaps, further her mammary development, but will not diminish the hirsutes. In this instance, a more total original mutilation would have been preferable, since removal of the other gonad before puberty would probably have prevented the virilization.

It is important to note that there was strongly female psychological orientation in this patient, who was

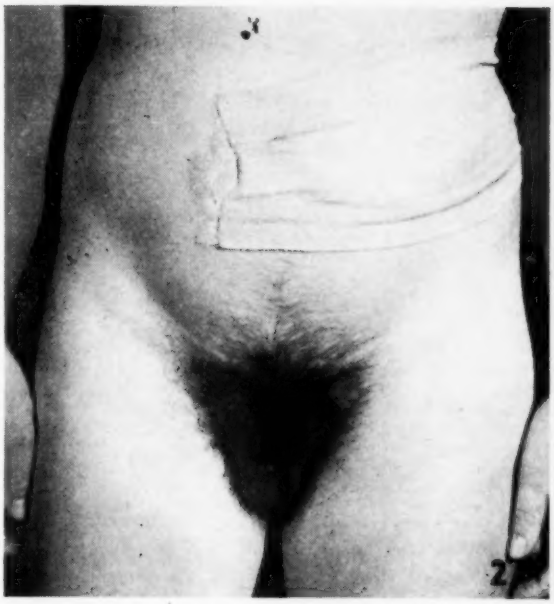

Fig. 2. Case 1 (M.E.). Note right scrotal swelling.

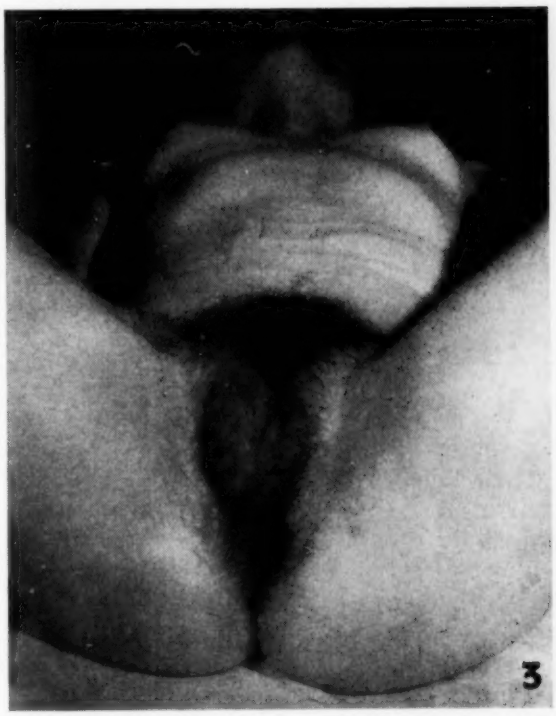

Fig. 3. Case 1 (M.E.). Pubis following amputation of penis and left gonadectomy.

male genetically, hormonally, gonadally and in respect of internal accessory organs of reproduction.

Case 2

L.R. European 28 years old. Despite the ambiguous nature of the external genitalia at birth, this patient was raised as a female. Throughout early childhood her inclinations were towards male pursuits. She preferred to climb trees and play male games, showed little interest in dolls or clothes and always felt strongly that she should have been a boy. At 16 years her voice became deeper, male sex hair appeared and the limb musculature began to develop in a male fashion. Menstruation did not appear and the breasts failed to enlarge.

She continued to live as a female, but still showed a preference for male company socially and females sexually. Her associates constantly suggested to her that she had been assigned to the wrong sex and advised her to undergo a 'change'. Recently the patient has become interested in a young girl and is considering marriage. This has stimulated her to come to Cape Town for 'rectification' of her sexual state.

On examination (Figs. 4 and 5), the scalp hair was seen to be cropped short in male style and there was moderate temporal hair recession; facial and body hair was profuse, with a typical male pubic escutcheon. The limbs were hairy and muscular; the breasts were almost non-existent. External genitalia consisted of a single urogenital sinus with a large phallus, but the perineal opening was so small that neither digital examination nor a hysterogram was possible. Cystoscopy was performed under anaesthesia (Mr. R. B. Watson) and a small vaginal opening was seen; dye displayed a small uterus (Fig. 6). No gonads were palpable.

A psychological appraisal was undertaken by Dr. W. A. Saffery and her orientation was regarded as strikingly male.

The diagnosis at this time was male pseudo-hermaphroditism. Taking into account her marked virilization (even to the extent of incipient baldness) and, particularly, her strongly male psychological orientation, the decision was made to convert her to full masculinity. The 17-ketosteroid excretion was then reported to be 134 mg. in 24 hours—a figure some 10 times normal. This finding completely refuted our previous diagnosis and indicated female pseudo-hermaphroditism (adrenogenital variety). Nevertheless, in view of her age and complete male orientation, the sexual conversion was proceeded with.

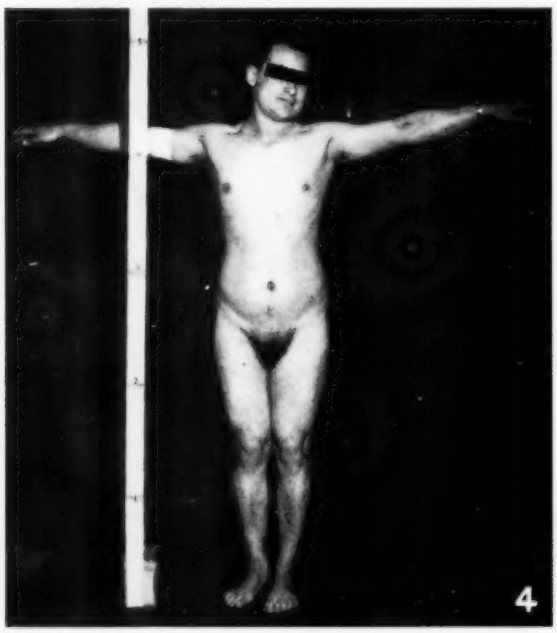

Fig. 4. Case 2 (L.R.). Note masculine habitus.

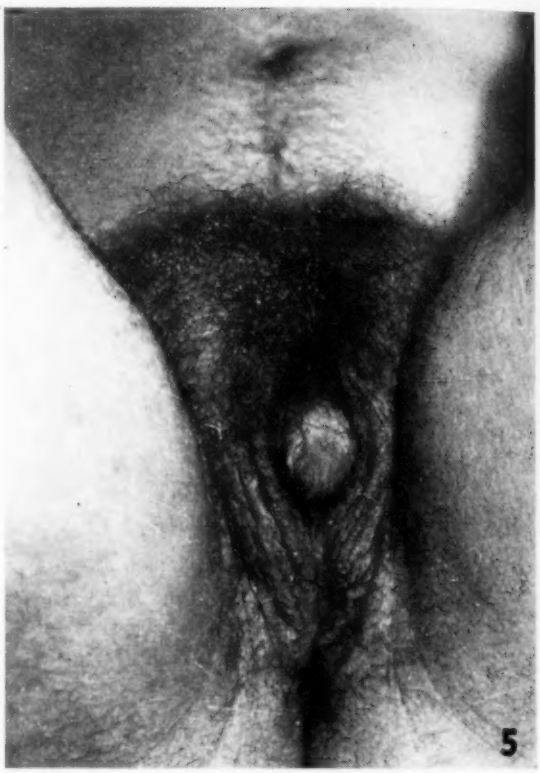

Fig. 5. Case 2 (L.R.). Appearance of external genitalia showing enlarged clitoris.

Professor James Louw and Mr. R. B. Watson found a very small uterus, well-developed fallopian tubes and gonads which, macroscopically, were not readily identifiable. Total hysterectomy and bilateral salpingogonadectomy was performed. Histology showed that the gonads were ovaries. Plastic masculinization with canalization of the phallus (to enable the patient to urinate standing) has been performed by Mr. N. Peterson.

Comment. It is most unusual for patients with the adrenogenital syndrome who are reared as females to show such striking male feelings. Money *et al.*[14] found in 38 hyperadrenocortical patients only one who exhibited bisexual interests. The overwhelming majority of patients adopt the gender role which is in accord with their assigned sex and sex of rearing (see later discussion).

If such a patient is diagnosed correctly at birth, treatment with cortisone will prevent all virilization and permit of a normal female existence, even including reproduction in later years. In this patient, at this stage, cortisone would serve no useful purpose and

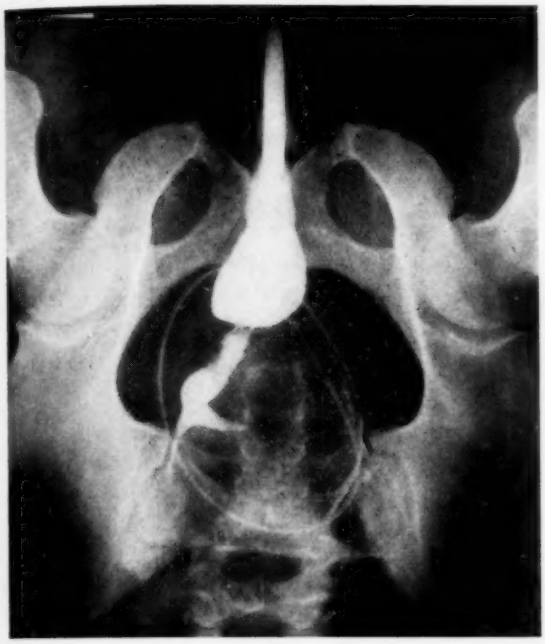

Fig. 6. Case 2 (L.R.). Injection of dye displays uterus and left fallopian tube.

future therapy must be aimed at consolidating her increased masculinity.

DISCUSSION

The Diagnosis in Hermaphroditism

There is no place for 'snap' diagnoses in the consideration of the ambisexual patient; those who appear to be members of one sex are often shown to belong to the other. Each patient requires careful examination of the external genitalia with roentgenological visualization of any orifice in the phallus or in the perineum; 17-ketosteroid excretion must be estimated; and where doubt is still present, laparotomy with bilateral gonadal biopsy may be necessary to establish the diagnosis with certainty.

Recently another valuable diagnostic tool has become available. Barr and his associates[1,2,15,16] have demonstrated that the nuclei of skin cells differ in males and females. In the latter the XX chromosome (or some attribute of it) is visible in 25-60% of cell nuclei as a small dark dot; in males such a mass is visible in less than 5% of cells. The same phenomenon may be seen in cells of the oral or vaginal mucosa and in polymorphonuclear neutrophil leucocytes.[6,7,15]

By means of this examination one may determine the genetic sex of the patient. Whatever the external appearance of the patient, one can ascertain whether the individual was destined genetically to be male or female. Thus, female pseudo-hermaphrodites with hyperadrenocorticism show female-type nuclear chromatin patterns regardless of the degree of virilization. Male pseudo-hermaphrodites show the male pattern, whilst true hermaphrodites may show one or the other. In practice the finding of a male skin-pattern excludes female pseudo-hermaphroditism and *vice versa*.

Correct Sex Assignment in the Neonatal Period

It has been shown that children begin to show gender awareness at approximately 18 months of age. By 2½ years a child has assimilated his role as a boy or a girl so thoroughly that a change of sex should not be made lightly.[13] It is therefore of prime importance to diagnose the type of ambisexual development early in life and to assign the child to that sex in which it will be least incapacitated.

It is a mistake to assume that the child must be raised in the sex which corresponds to its gonads. For instance, the most extreme form of male pseudo-hermaphroditism is completely female in body-build and external and internal genital development. It is patently ridiculous to rear such a patient as a male.

Where possible there should be an attempt to forecast the changes which will ensue at puberty. Case M.E. above illustrates the results of failure to consider this factor—the unhappy sequelae of pubertal virilization in this patient might have been avoided. A forecast of this nature requires accurate diagnosis of the underlying aberration and justifies procedures such as laparotomy and gonadal biopsy.

A very important consideration is the structure of the external genitalia. If these are so strongly male or female in type as to defy surgical reconstruction, then the child should be assigned to the sex which it resembles. Future therapy must be aimed at furtherance of development in the direction of the assigned sex. Where no such marked predominance is found, other factors may be considered, e.g. gonadal type or hormonal preponderance.

The outstanding exception to the above precepts is the condition of hyperadrenocorticism producing female pseudo-hermaphroditism. This state can usually be diagnosed with certainty in very early life and Wilkins[23] has shown the remarkable restoration to normal femininity which follows cortisone therapy.

Considerations in Older Patients

Many patients are seen in later life after they have already been reared in one or other sex. In this group the gender role* has become well established, although ambivalence may exist. Here, generally, psychological considerations are the most important. Full and careful psychological appraisal is necessary before any decision can be taken.

Case L.R. above illustrates this point well. This patient was female genetically, gonadally and by up-

* By gender role is meant 'all those things that a person says or does to disclose himself or herself as having the status of a boy or man, girl or woman, respectively. It includes, but is not restricted to, sexuality in the sense of eroticism. A gender role is not established at birth, but is built up cumulatively through experiences encountered and transacted . . .'[13]

bringing; yet her psyche was developed in a very strong male direction. This was the consideration which prevailed upon us to alter her status to that of a male.

In the vast majority of hermaphrodites the psyche has been shown to develop in accordance with the assigned sex and sex of rearing, rather than the structure of the internal or external genitals, or the gonadal or chromosomal sex. It is therefore exceptional to find a hermaphroditic subject reared in one sex whose psychological orientation is that of the opposite sex. Money *et al.*[14] found only 4 of a series of 76 hermaphrodites in whom psychological ambivalence was detected. The remaining 72 all developed mentally in accordance with the sex to which they had been assigned. This small percentage of patients showing lack of psychosexual concordance is, perhaps, no higher than the percentage of 'normal' people who are found to show psychosexual ambivalence.

CONCLUSIONS

The incidence of bisexual anomalies at birth is high. Differentiation of the underlying defect is usually difficult and the 'spot' diagnosis is frequently incorrect. At a very early age every attempt must be made to establish the correct diagnosis; at the same time a firm decision must be made with regard to assignment of the child to one or other sex; such assignment should generally be made on the basis of external genital anatomy. Where this is completely ambiguous, other factors may be considered. Once this decision has been made, artificial devices may be employed to further development in the 'chosen' sex, e.g. gonadectomy or hormonal therapy.

It is of the utmost importance that the true facts should be withheld from both patients and parents in those instances where the choice of sex differs from genetic or gonadal sex. In case 1, for example, the disclosure of the patient's true sex could only occasion distress.

The psychological state of affairs in older patients must be very carefully appraised before any decision is taken about allocation to one or other sex.

In the consideration of intersexual subjects, the following aphorisms may be of value:

1. A fully-descended gonad is a testis or an ovotestis; it is never an ovary. Female pseudo-hermaphroditism may thus be ruled out.

2. A male skin-chromatin pattern also rules out female pseudo-hermaphroditism; a female pattern excludes male pseudo-hermaphroditism.

3. Hence, female skin-sex with a descended gonad indicates true hermaphroditism.

4. Female pseudo-hermaphrodites with the adrenogenital syndrome—and *only* these—always have a raised output of 17-ketosteroids.

5. Female pseudo-hermaphrodites, if treated, may well be fertile; male or true hermaphrodites cannot become parents.

6. Precocious puberty of male type in an intersexual individual indicates hyperadrenocorticism with respect to androgen (i.e. female pseudo-hermaphroditism).

7. Those intersexuals who exhibit neonatal episodes suggesting Addisonian crises are female pseudo-hermaphrodites with adrenocortical overactivity.

8. Every 'male' without palpable testes must be suspect, *whatever the state of his genitals.*

9. Every 'female' who does not have a menarche must likewise be suspect.

SUMMARY

The current views on intersexual states are briefly summarized. Two case-reports are presented to illustrate some of the difficulties in diagnosis and to emphasize the unfortunate sequelae of incorrect diagnosis and sex assignment. A plea is made for thorough investigation of cases at an early age and for firm decisions with regard to rearing. Some of the important factors which should influence one's decisions are reviewed.

We are pleased to thank Professor F. Forman, Professor J. F. Brock and Mr. D. J. du Plessis for advice and criticism, Professor James Louw, Dr. Helen Brown and Dr. M. Horwitz for permission to report the patients, Dr. S. Berman and Dr. W. A. Saffery for the psychological appraisals in the two patients and Mr. B. Todt for the photographs. Thanks are also due to the house-physicians, Dr. D. Stein, Dr. C. J. Blignaut and Dr. V. Dubowitz for their interest and cooperation.

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CARCINOMA OF THE BLADDER

A REVIEW OF TREATMENT*

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Johannesburg

While on a recent visit to Great Britian and the Continent of Europe I had the opportunity of visiting numerous clinics. I was impressed by the large number of bladder tumours I saw, the incidence of which appears to be greater than in South Africa. In Manchester the Christie Hospital specializes in the treatment of cancer and consequently cases are referred to them from all over England; moreover, the I.C.I. maintains a large dye works in Manchester, and bladder tumours amongst dye workers being notifiable, routine cystoscopy and a complete follow-up is possible.

The treatment of bladder tumours differed in almost every clinic I visited, and if one refers to the literature during the last 25 years innumerable methods of treatment of bladder tumours have been advocated, most of which have proved very disappointing. To what can this be attributed? The answer is that cases are referred for treatment too late; either the patient does not seek advice or the patient's doctor is foolish enough to prescribe some form of treatment for the haematuria without establishing a diagnosis.

The success of treatment depends on 3 factors: (1) Degree of invasion or infiltration, (2) type of tumour, and (3) metastases.

1. *Degree of Invasion*

In 1946 Jewett published a most interesting paper on the relation of the depth of penetration of the bladder wall to the incidence of local extension and metastases. This was a review of a large number of cases of infiltrating tumours of the bladder upon which autopsies had been performed at the Johns Hopkins Hospital during the previous 25 years. Jewett divided the cases into groups viz.

Group A—submucosal infiltration.
Group B1—superficial infiltration of muscular layer.
Group B2—deep infiltration of muscular layer.
Group C—perivesical infiltration.
Group D—lymphatic and distal metastases.

He showed that group-A cases are theoretically 100% curable. Group-B cases are 88·8% curable, but as soon as the tumour reaches the perivesical tissues the curability drops to 26%. This is not surprising when one studies the variation in size and distribution of the lymphatics of the bladder wall, so beautifully demonstrated by Powell. Of the group-C cases 58% had glandular and distant metastases, 37% had lymphatic-gland involvement only, and in only 7·7% with perivesical infiltration were there no glandular of distant metastases or, to put it more forcibily, if perivesical infiltration has occured 92% have metastases. In cases with glandular and distant metastases 26% showed no fixation to surrounding structures; it thus follows that if any fixation exists the percentage of curability is extremely low. Tumours of the anterior and lateral walls of the bladder metastasize earlier than those of the posterior wall.

2. *Type of Tumour*

The following classification introduced by Dukes and Masina is simple and easily followed:

(*a*) Simple transitional-cell papilloma
(*b*) Malignant tumours:

(i) Papillary transitional-cell carcinoma
(ii) Solid transitional-cell carcinoma
(iii) Transitional-cell carcinoma with metaplasia
(iv) Pure squamous-cell carcinoma
(v) Pure adenocarcinoma
(vi) Anaplastic spheroidal-cell carcinoma

The histology of these different types of tumours is as follows:

(*a*) *Simple transitional-cell papilloma.* Central vascular connective-tissue cord covered by transitional epithelium, which may be several cells deep, sharply defined, regularly arranged, with basic membrane intact, and with no sign of invasion or infiltration of the vascular core.

(*b* i) *Papillary transitional-cell carcinoma.* Similar in structure to (*a*) but the cells have a disorderly arrangement, irregularity in shape and size of nuclei, sometimes a typical mitosis, and a tendency to metaplasia. Invasion and infiltration seen as a breaking through the basement membrane and clumps of carcinoma cells in the connective-tissue core.

(*b* ii) *Solid transitional-cell carcinoma.* Most bladder growths in the early stages show a papillary pattern; as infiltration takes place they become more solid, the cells being arranged in more compact clusters. The less malignant tumours tend to remain papillary in structure.

(*b* iii) *Transitional-cell carcinoma with metaplasia.* A peculiarity of bladder tumours is the tendency of the epithelium to undergo metaplasia with the formation of squamous or glandular epithelium in those tumours of a high grade of malignancy. Scattered islands of squamous epithelium often resemble the cell nests of a cutaneous epithelioma, and glandular epithelium may present a typical picture of an adenocarcinoma.

(*b* iv) *Pure squamous-cell carcinoma* is a rare tumour and is almost invariably associated with a previous leucoplakia. It is probable that these tumours are less malignant than transitional-cell carcinoma with squamous metaplasia.

(*b* v) *Pure adenocarcinoma.* While metaplasic glandular eptihelium may be present in a bladder carcinoma, a pure adenocarcinoma is rare and arises either from the remnants of a urachus or in epithelium which has previously undergone glandular metaplasia, as in

* A paper presented at the South African Medical Congress, Pretoria, October 1955.

extroversion of the bladder; otherwise the adenocarcinoma is probably an extension or metastasis from the bowel.

(*b* vi) *Anaplastic spheroidal cell carcinoma.* Tumours of undifferentiated spheroidal or polygonal cells are very invasive, grow rapidly, and metastasize early. The histology varies greatly and is sometimes indistinguishable from sarcoma.

3. *Metastases*

This classification is based on autopsy findings and microscopic examination of pathological material removed by partial or total cystectomy. In what way can we apply the knowledge so gained to our clinical investigation? Initially the diagnosis is established by cystoscopy, and where possible the following observation should be made:

(*a*) *Gross characteristics of the tumour*

(i) Whether single or multiple, and exact situation
(ii) Size
(iii) Shape
(iv) Appearance of surface-villous or cauliflower, and whether sloughing
(v) Appearance of surrounding mucosa
(vi) Whether bladder distension causes bleeding

(*b*) *Biopsy.* This should always be taken as complete as possible, including the base of the tumour and as much musculature as possible. A resectoscope punch or biopsy rongeur should be used.

(*c*) *Bimanual examination.* This is of paramount importance. While a large papillary tumour may be palpable any degree of infiltration and fixation indicates a hopeless prognosis.

(*d*) *Cystogram.* This is of value, for a series of pictures may indicate more accurately the size of the tumour and the degree of invasion of the bladder wall.

(*e*) *Intravenous pyelography.* This should always be done, for dilatation of one or other ureter indicates infiltration of bladder wall.

TREATMENT

Assessment of Case

While the degree of infiltration of the bladder wall is the most important factor in prognosis, it is obvious that the less differentiated and more malignant types of growth infiltrate and metastasize more rapidly. In recent articles Chapman and others have indicated the clinical significance of biopsy examinations, and statistics show that more drastic methods of treatment have to be adopted in tumours of a high degree of malignancy.

Methods of Treatment

These include the following:

1. Transuretral fulguration
2. Open fulguration and electro-coagulation
3. Diathermy excision and implantation of radon seeds, needles of radium or radioactive cobalt, tantalum wire, or gold grains
4. Intracavitary radiation
   (*a*) Solid central source, e.g. radium or cobalt
   (*b*) Radioactive solution, e.g. sodium bromide or cobalt
5. Radioactive gold solution
6. Partial cystectomy or segmental resection
7. Complete cystectomy, simple or radical
8. Deep X-ray therapy.
   (*a*) Contact therapy.
   (*b*) Deep X-ray therapy (250-500 kv. machines)
   (*c*) Supervoltage X-ray therapy—1-million to 20-million-volt machines

Transurethral fulguration. All simple papillomata should be treated by this method and theoretically should be 100% curable. Many urologists advocate diathermy resection and electro-coagulation in malignant tumours of grade A and grade B1 according to Jewett's classification. At the Mayo Clinic, of 1,222 cases, 961 were treated by transurethral resection and fulguration, with 88% 5-year cures. Milner and Flocks using conservative methods quote the following percentages of 5-year cures in cases classified by Jewett's method:

	A	*B1*	*B2*	*C*
Milner	70%	57%	23%	1 case
Flocks	77·4%	56%	39%	1·6%

Open fulguration and electro-coagulation. This is, as a rule, only carried out in very large tumours where the size and situation make transurethral resection difficult or impossible, or as a preliminary step before implanting radium etc. Special precautions have to be taken to prevent tumour implants developing in the wound.

Implantation of radon seeds. This treatment has been used for many years and the results are very encouraging. Radon seeds of a strength of not less than 1 millicurie are implanted 1 cm. apart encircling the tumour and in the base of the tumour; this may be done transurethrally, but for accuracy the suprapublic route is preferred. The bladder should then be closed, as radon seeds are not removed. The following are published 5-year-survival results (again according to Jewett's classification):

PUBLISHED RESULTS OF 5 YEAR SURVIVALS

	No. of cases	*A*	*B1*	*B2*	*C*
Poole Wilson (unclassified)	104		49%		
Poole Wilson (classified)	62	71%	50%		11·6%
Milner (unclassified) ..	90		22%		
Barringer	221	52%		23·6%	
Millen	117	63%		21%	
Dean and Balfour ..		54%		14%	
Emmett & Mayo Clinic	118		29%		

The figures from the Mayo Clinic are of particular interest, because radon-seed implantation was not favoured as a method of treatment and was only used when other methods of treatment were inapplicable, and yet there was a 5-year survival rate of 29%. Emmett states that 'as a result of this study we feel that resection plus inplantation of radon seeds merits more frequent use than has been accorded in the past'. In use gold grains are similar to radon seeds, but they loose their activity more rapidly, the 'half-life' being 2·7 days compared with the 3·8 days of radon seeds.

Implantation of radium, radio-active cobalt, or tantalum wire. These substances are favoured by many surgeons. Implantation has to be carried out suprapubically, and it is often difficult owing to the curvature of the bladder wall to place the needles or wire accurately. The dosage is calculated from X-ray photographs. The main disadvantage of this form of treatment is that the bladder cannot be closed, although it is possible to close the bladder and bring the attached threads and a catheter through a urethrostomy opening. Few statistics have been published: Jacobs quotes 110 cases with a 34% 5-year survival rate, Kligeman 91 cases and 38% survival, Catgill 20% survivals.

Intracavitary radiation. Specially constructed balloon catheters have been used for this purpose. Generally speaking the results have been disappointing. Sufficient penetration cannot be obtained to destroy growths of B1 & B2 group, without causing severe bladder irritation and fibrosis. It is probably most useful in generalised papillomatosis. The handling of radioactive solutions such as Radioactive Sodium and Bromine requires elaborate apparatus and the results have been disappointing.

Radio-active liquid gold. This method is most suitable for papillomatosis, but it is impossible to estimate the dosage and the method is unsuitable for infiltrating tumours.

Partial cystectomy or segmental resection. This method of treamtent has been carried out for many years, and is still advocated by many surgeons. The results have been disappointing and published results do not compare favourably with other methods of treatment. Five-year survival rates: Milner 28%, Mayo Clinic 27%, Jewett 23·7% (Groups A and B 68%, groups B2 and C 7%), Kligerman 22%, Riches 21·7%, Jacobs 20%. It is generally accepted that partial cystectomy should be carried out in all tumours of the vault or upper half of the bladder.

Total cystectomy. During the last decade there has been a wave of enthusiasm for the operation of total cystectomy. At the beginning the mortality rate was very high, in some instances over 50%; with more experience and improved technique the mortality rate has been reduced as low as 5%. Inasmuch as most surgeons reserve this operation for cases unsuitable for any other form of treatment, viz. penetrating tumours (when the penetration has reached the perivascular tissues 93% have glandular or distal metastases) the results have not been very encouraging. Published statistics of 5-year survivals: Mayo Clinic 19%, Kerr and Colby 10%, Riches 9%, Jewitt 19%, but all show severe renal damage secondary to the ureteric transplant. Marshall, who extended the operation to include all grades of carcinoma and so-called simple papillomata, had even more disappointing results, only 26% 5-years survivals. More recently radical cystectomy, including the prostate and all the pelvic lymphatics, and in the female panhysterectomy, has been advocated. Few statistics of this procedure are available; Emile Sayegh published a review of 106 cases which is of interest: Of 21 cases which had palliative uretero-colic amastomosis, 18 died within 18 months and only 1 survived 2 years, and then died. Of 23 cases which had bilateral uretic transplant and total cystectomy, 18 died during the first year and only 1 survived longer than 2 years. Of 62 patients who had radical cystectomy and pelvic adenectomy, 8 or 13%, have survived for longer than 3 years, with no sign of recurrence.

The enthusiasm for total cystectomy is on the wane because of poor results, renal damage following ureterocolic amastomosis, and electrolytic imbalance. The following types of lesion, however, are best treated by cystectomy: (1) Leucoplakia of the bladder, (2) diffuse papillomatosis, (3) irradiation cystitis and uncontrollable bleeding following irradiation, and (4) extensive superficial lesions. Some surgeons advocate cystectomy as a palliative measure, it will not prolong the patient's life, but may relieve his suffering.

Deep X-ray therapy. For contact therapy the lesion has to be exposed by open cystostomy and irradiated by direct X-ray therapy by means of a sterilized applicator. It is thus a 'single shot' treatment, and there are few lesions which cannot be more adequately dealt with by other forms of irradiation. *Deep and supervoltage X-ray therapy* have usually been employed in the treatment of invasive carcinomata which were too advanced for any other form of treatment. The results, however, have been very encouraging. The following are the 5-year survival results of deep X-ray therapy at the Christie Hospital and Holt Radium Institute, Manchester:

	No. of cases	*% of total no. of cases* (289)	*5-year Survival*
Radical X-ray Therapy			
Diffuse Papillomatosis ..	17	6%	8 (47%)
Gross infiltrating carcinoma	66	23%	17 (25%)
Palliative X-ray Therapy			
Gross infiltrating carcinoma	48	17%	3 (6%)

Bloomfield, at the United Sheffield Hospitals, after deep X-ray therapy had 14% alive and apparently free of tumour for 2 years. Better results followed the use of supervoltage therapy, 44% being alive after 2 years.

CONCLUSION

From the above analysis the following conclusions may be drawn regarding the best methods of treatment of bladder tumours:

Simple papillomata and grade-A and grade-B carcinomata should be treated by fulguration, resection and electro-coagulation, grading of the tumour being determined by cystoscopy, a carefully-taken biopsy, and bimanual examination. In grade-B1 and grade-B2 tumours the best results are obtained by resection and radon-seed implantation. Sequental resection should be reserved for tumours of the vault or upper half of the bladder. Should cystectomy be contemplated, it should be a radical cystectomy with complete pelvic adenectomy. The radio-active isotopes can be used effectively where radon seeds are not available. Cobalt needles and tantalum wire will no doubt be used extensively in South Africa. There is undoubtedly a place for X-ray therapy in the treatment of vesical carcinoma, more particularly supervoltage X-ray therapy, and the results would be considerably better if this treatment were not reserved for the hopelessly inoperable cases.

THE SURGICAL MANAGEMENT OF ULCERATIVE (STASIS) DISEASE OF THE LOWER EXTREMITIES*

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There is a great deal of confusion about varicose veins and varicose ulceration of the lower extremities. The purpose of this paper is twofold: firstly to attempt to clarify the present-day concept of varicose ulceration of the lower extremities, and secondly to outline a surgical treatment of varicose ulceration especially for the ulcerative stasis disease of the lower limbs. This stasis disease is also referred to as gravitational ulceration and as ulceration of the post-phlebitic syndrome. It is proposed to describe briefly in tabulated form the clinical features of the different pathological states that are associated with varicosities. Clinically varicose veins can be distinguished as (1) primary and (2) secondary. The clinical features are classified under these headings as follows:

Primary	*Secondary*
Etiology	
Heredity. Pregnancy. Occupational stresses, of standing.	Usually secondary to deep-vein thrombophlebitis, especially where anticoagulant therapy not or inadequately carried out. Occasionally idiopathic.[1]
Oedema	
Minimal; swelling confined mainly to bulging of varicosities.	Most prominent feature, especially at the end of a day's work requiring standing.
Ulcers	
Malleolar in site, surrounded by darkish pigmentation and often eczema; usually single, and indurated cellulitis not a usual feature.	Not necessarily malleolar. Irregularly placed, multiple, often situated more proximally and associated with a greater amount of induration, eczema and cellulitis. Indurated cellulitis a prominent feature. Fungus infection of skin common.
Symptoms	
An ache on standing relieved by activity and walking. Nocturnal cramps. Signs and symptoms of complications, e.g. superficial phlebitis, pain of the ulceration and secondary infection. Distribution of varicosities conforms usually to the anatomical pattern of the long and lesser saphenous veins.	Bursting feeling and ache on standing. Severer symptoms generally. Intractable and recurrent painful ulceration. Repeated attacks of cellulitis in indurated areas, apart from ulceration of skin. Nocturnal cramps usually more severe and more frequent. Varicosities conform to irregular pattern with the presence of 'blow-outs' and feeder veins from ulcerative and eczematous skin.

PATHOLOGICAL CHANGES

Both primary and secondary varicose veins are complicated by venous stasis disease, resulting in severe skin changes of the distal parts of the lower extremity. It is generally accepted that the underlying cause of varicose veins in the absence of any deep-vein disease is an intrinsic defect in the vein wall of the saphenous system itself, in addition to the occupational factors which may keep a patient standing for long periods. As indicated, swelling is not a prominent feature in the primary varicosities, although ulceration and skin changes do occur. Nor is indurated cellulitis a prominent or regular feature. Radical and efficient removal of the offending varicosities, blow-outs and feeder veins from the affected skin areas results in satisfactory and lasting cures for many years in the great majority of cases.

The outstanding feature, however, of the ulcerative stasis disease of the lower extremities, which is usually secondary to deep-vein disease (from any cause), is the invariable tendency to swell, to produce incompetent superficial varicose veins, and to produce the severe crippling disabilities of varicose ulceration, eczema and indurated cellulitis[2]. These changes affecting the skin and subcutaneous tissues of the stasis area occur on the medial and lateral aspects of the distal third of the leg. The oedema is ascribable to lymphatic-vessel disease and to venous insufficiency, either or both factors being present in varying degrees in individual cases. Unless repeated episodes of inflammation have occurred, the lymphatic element ordinarily diminishes with time. It is severest in the early period following the acute thrombophlebitis to which it is an accessory. The venous element of the stasis arises either from permanent occlusion of the deep veins of the extremity or the tendency for reflux venous flow in a vein which has been rendered valveless by the original infection. The varicose veins which are characteristic of stasis disease appear in response to the general increase in the venous bed and pressure secondary to deep-vein disease of the legs. Venous stasis accompanied by an appreciably increased vein-pressure results in oedema. If one considers the end stage of deep-vein thrombosis in a deep principal vein as a stiff tube having thick walls of connective tissue and non-functioning valves, one can easily appreciate the considerable capillary pressure which occurs in the lowest part of the leg when the 'vein pump' is unable to counteract the hydrostatic pressure in the valveless principal veins. The secondary varicosities forming in such a limb are the subcutaneous component of the developing collateral venous system. Inasmuch as these veins are unsupported by fascia and muscle pressure they quickly dilate, rendering the valves incompetent, and they become themselves reflux venous channels. It is important to appreciate that these superficial varicosities secondary to deep vein disease are incompetent, and their presence merely aggravates venous stasis. It is therefore logical to ablate such varicosities, contrary to the generally-held

* A paper read at the South African Medical Congress, Pretoria, October 1955.

view that no limb which has been the site of deep-vein thrombophlebitis should have such varicosities removed because this will lead to a greater degree of oedema. This erroneous belief is based on the concept that these secondary varicosities are the only channels for the return of venous blood from the lower extremities. These secondary veins serve no such function; they only help to perpetuate the pathological processes. There are other return channels through which venous return can efficiently take place, viz. muscular veins, recanalized diseased deep veins, and the capsular veins of the knee joint.

The skin changes which appear both in stasis disease and in primary varicose veins are characteristically located in the same areas, with minor difference in their situation. These areas are the medial aspect of the ankle above the malleolus in the course of the long saphenous vein system, and the lateral aspect at about the same level in relation to the lesser saphenous system. To explain this uniformity of the affected area it must be postulated that some peculiarity exists in the structural anatomy at these points or in the pressure relationships within the veins, which may be responsible for the changes in the skin and subcutaneous tissue. If such a peculiarity can be worked out a method might become available for the permanent healing of stasis ulceration. The probable etiology of the ulcers which appears in these areas is the presence here of the greatest number of communicating veins between the deep venous system and the superficial channels. This reasoning has been applied by Linton[3] in developing a method of individually ligating these branches. The presence of these imcompetent perforating veins has caused a number of workers to reduce venous stasis by means of uniform external pressure.

Besides the presence of the perforating veins in the area of ulceration, the character of the tissue changes which precede the breakdown of the skin must be considered. Changes advanced enough to make a return to normal unlikely must be recognized as a factor determining the need for total eradication of the diseased skin and subcutaneous tissues of the areas. In attempting to treat these conditions surgically one must take into consideration pathological processes that take place. This surgical therapy includes the removal of all the damaged skin and subcutaneous tissue together with the underlying fascia; the long and lesser saphenous systems, and the local perforating and feeder veins. The irreversible character of the tissue changes locally seems to warrant this radical approach.

The results obtained by this surgical treatment, and the observations on the secondary stasis changes and the anatomy of the perforator and feeder veins at operation, are described below.

CLINICAL MATERIAL

The surgical management of these cases of stasis ulceration of the lower limb is based on 1,000 cases of primary and secondary varicosities operated on over the past 10 years both in hospital and private practice. Of these 1,000 cases, 200 phlebectomies were performed on patients with a definite history of previous deep-vein involvement and showing signs of the typical post-phlebitic syndrome. In these 200 cases, 30 skin grafts were combined with the phlebectomy operation.

DETAILS OF TREATMENT

The preoperative management follows the conventional methods and is directed towards elimination of infection in and around the ulcerated area, complete control of the oedema of the foot and leg and the re-epithelization of the ulcerated area. Bed rest and elevation of the limb is absolutely necessary to achieve these aims. No ulceration will heal rapidly or completely without bed rest. The parenteral administration of antibiotics has reduced the morbidity considerably and relieved the pain of these skin conditions. Control of a commonly superadded fungus infection is absolutely essential. Repeated moist dressings of saline with and without antibiotic solutions are applied together with solutions of aluminium acetate. For the past year we have had most favourable results in the treatment of infected ulceration and skin lesions with a solution of streptomycin (1 g. to 100 c.c.) applied as a moist dressing and kept constantly moist with repeated applications or the addition of the solution to the dressing *in situ*. This moist dressing must be applied to the ulcer and infected area only, otherwise maceration of the healthy surrounding skin is likely to take place.

The severity and extent of the cutaneous and subcutaneous fibrosis and the extent of the varicosities is determined by palpation in the erect position after the oedema has subsided.

As already indicated, in the majority of cases of ulceration due to primary varicosities a complete and efficient eradication of the long and lesser saphenous systems, the secondary cutaneous branches, the perforators, and feeder veins, is all that is required. This eradication is really a phlebectomy of all the superficial varicosities.

It is not intended to describe in detail the surgical technique employed for the 1,000 cases over the past 10 years, but to discuss general principles. This procedure is a major one, not to be carried out as an out-patient procedure. The patient is carefully examined to exclude any other vascular problem such as obliterative arterial disease or arterio-venous fistula. We do not utilize any of the tests to detect perforator branches, because the technique is so radical that all these are extirpated.

Proper pre-operative preparation of the skin of the lower limb including the lower half of the abdomen is carried out.

General anaesthesia is employed. If a bilateral operation is required, one leg at a time is operated on with a week's interval. Careful marking of all superficial varicosities is carried out before anaesthesia. Bonney's blue is the best dye to use—others are inclined to run with cleaning and swabbing of the leg. All main trunks, secondary branches, and especially the feeder veins from ulcerated areas, are carefully marked in continuity.

High ligation of the long saphenous through a vertical groin incision is performed, exposing it with its tributaries at the saphenofemoral bulb. The tributaries are so often anomalous that they do not conform to the anatomical text-books; for example, a double saphenous is not uncommon and branches vary in number from 1 to 6. Extraluminar stripping of the main saphenous trunk is carried out with the aid of an extraluminar Mayo stripper, down to the ankle and even beyond to the foot. This will entail stripping

behind healed ulceration. We have found the intraluminar stripper quite inadequate for this purpose because of anatomical anomalies.

The radical excision of the secondary branches must be carried out by incisions—usually lengthy ones over these branches themselves, for we have found the use of intra- or extraluminar stripping techniques for these branches to be unsuccessful. This phlebectomy may be tedious, but the results justify the labour. These secondary branches invariably reveal a perforator communicating with the deep trunk. Multiple ligation along the course of these branches is not sufficient as such a technique could quite easily miss these perforators.

Excision of feeder veins from the ulcer-bearing area is absolutely necessary; otherwise recurrence of ulceration is sure to follow. These feeder veins are also attacked through a direct incision in the length of the limb and up to the edge of the healed ulcer.

Post-operative care includes supportive elastoplast type of bandage of the whole limb (over cotton bandaging; otherwise zinc-oxide sensitivity with severe results has been known to occur). Over the stripped skin area and below the adhesive elastic bandages special elongated dressings are placed over the length of the limb to prevent post-operative effusion of blood and haematoma. The patient is made to get out of bed the following day and walk. Repeated muscular activity is also encouraged while lying in bed.

For secondary varicosities combined with skin ulceration and other skin changes, the same pre-operative treatment is carried out and the same technique of varicose vein ablation is used, but in addition attention is directed locally towards the healed ulcer and surrounding skin. It is carried out either in one stage combining the phlebectomy and the skin grafting, or in two stages, when the phlebectomy is done initially and thereafter the skin grafting carried out at a secondary operation. The two-stage technique has the advantage that in some of the cases the original phlebectomy has been sufficient to maintain a good result with the ulcer and skin lesions. If grafting is continued with at the initial operation, the ulcer area is excised widely enough to reach good skin. The subcutaneous lesions, which are densely fibrosed and often contain small abscess-like areas filled with cloudy fluid, is removed in a single tissue-block, taking with it all the deep fascia adherent to its under-surface.

From 1 to 6 veins of major size have been found in cases perforating the fascia in this area to communicate between the subcutaneous and deep veins. These are clamped and ligated and a split-thickness skin graft, taken usually from the antero-lateral aspect of the opposite thigh, is used to cover the defect after complete haemostasis has been effected. The graft is meticulously sutured in place with absorbable gut to the edge of the healthy skin. The graft is applied directly on the muscle bed and if a hollow exists in the graft bed, sutures are applied to the middle of it to this muscle bed to obliterate dead spaces as far as possible. Proper supportive post-operative presure-dressings also help to achieve this. Post-operative antibiotics are always employed as well as ascorbic-acid tablets in massive doses. Bed rest is essential until the first dressing is removed, usually 7-8 days after grafting. As these sutures are absorbable there is no necessity to remove them. When the graft has healed completely, a progressive regime of graded ambulation is instituted. All the graft operations performed (30) have healed particularly well, with only one immediate failure. The success of these grafts is striking and one explanation offered is the efficient lymphatic bed present.[4]

PATHOLOGICAL CHANGES AND LOCAL ANATOMY DETECTED IN THE STASIS AREA

Complete removal of the stasis area in one skin-block together with its deep fascia has provided an opportunity to observe the pathological anatomy and tissue changes which are present. In all our cases 1-6 venous channels have been noted, passing through the deep fascia between the subcutaneous tissue and subfascial veins. Within the excised tissue itself these are large-walled tortuous venous channels, which tunnel the densely fibrous tissue. The number and size of the communicating veins diminish strikingly at the border of the stasis area so that, although there are many veins within the removed specimen, there are much fewer at the edge of the excision. The cut surface of the skin specimen is fibrous and wet with liquid fat and very often milky fluid. Cultures of this material are sterile.

POST-OPERATIVE CARE OF OPERATED LIMB

Recurrences of ulceration have been observed with this grafting technique, and one has noted that this has occurred in those patients who live too far away to return for constant and repeated follow-up examination, and in these subjects oedema has been allowed to recur. It is our experience in 200 cases already referred to that eradication of all superficial varicosities will not remove the pathological process that results in oedema. It is wrong therefore to promise any patient that swelling will be cured. If oedema still persists then it must be effectively and rigidly controlled. We advise elastic stockings, elastic bandages, or both. If elastic stockings are used they must be fitted to the other normal leg, or to the affected limb when all oedema has subsided, after a night's rest in bed with the foot elevated. The stockings must be made of heavy two-way stretch material and must cover the heel, leg and lower thigh. They can be made of either nylon or cotton. Where occasionally one stocking will not control the oedema then the patient should wear two. In exceptional cases an elastic bandage may have to be applied over the elastic stocking. Some may prefer the stocking over an elastic bandage.

Patients are taught by means of an instruction sheet the care of feet and legs, cleanliness, supportive bandaging to eliminate oedema, not to stand excessively, and to indulge in a 2-hour rest during each day if this is possible. The patient is also instructed to keep the foot of his bed permanently raised 6-9 inches.

Mention has been made of indurated cellulitis in stasis disease of the lower extremities. This is often a severely painful and recurring condition, characterized by acute cellutitis, severe local pain and a dense 'pancake' of stasis change in which all of the pathology of this condition exists without ulceration of the skin. For these conditions we have applied the same operative treatment as outlined for stasis ulceration, carrying the graft to normal skin.

CLINICAL RESULTS

Thirty grafts which have been applied by this method in 26 patients have been followed up for periods of 2 years. The results are summarised in Table I.

TABLE I. CLINICAL RESULTS OF GRAFTING

	No. of Grafts	*No. of Patients*
Number of failures	8	8
Number successful	22	18 *

* In 4 patients grafts were performed bilaterally.

TABLE II. POST-OPERATIVE OEDEMA IN RELATION TO GRAFTING

Grafts	*Post-operative Control of Oedema*	
	Good	*Poor*
Successful 22 in 18 patients ..	17	5
Failures 8 in 8 patients	1	7

COMMENT

It is hoped that this paper has indicated two definite groups of varicose veins, viz. primary and secondary, which both cause venous stasis disease. The secondary, however, is more severe and the accompanying stasis ulceration more marked. Stasis ulceration can be considerably relieved by a radical phlebectomy, with or without a wide excision of the ulcer-bearing area, to which a skin graft is applied. The incompetent communicating veins in the stasis area are likewise completely ablated. Failures do occur but only when the venous stasis is associated with secondary varicosities. These failures are most frequent in patients with marked and persistent oedema which is either uncontrolled by efficient supportive stockings or bandaging, or controlled with difficulty.

The techniques of ligation of the popliteal vein as recommended by Bauer,[5] or ligation of the superficial femoral vein as described by Linton,[6] are not included in the operative measures described here.

This work is based on 1,000 cases of primary and secondary varicosities operated on. Of these 200 were secondary varicosities for which a radical phlebectomy was carried out. In the latter group 30 cases were combined with grafting.

SUMMARY

The severity of stasis changes in the lower limbs leading to skin ulceration and other skin lesions is discussed on an etiological and pathological basis.

A radical phlebectomy of the long and lesser saphenous systems together with the excision of secondary branches, perforators and feeder veins is briefly described.

This operation together with excision of irreparably diseased ulcer-bearing skin is offered as a means of salvaging a great number of these severely crippled subjects.

The majority of ulcers resulting from primary varicose veins will heal with efficient eradication of these veins alone.

Failures with the more intractable ulceration associated with the group of secondary varicose veins do occur.

Operation to remove varicose veins secondary to deep-vein disease can be carried out without any further oedema or damage occurring in the affected limb. Two hundred such cases have had these secondary veins removed with satisfactory results. This dispels an old erroneous belief that it is unwise to remove such varicosities. It must be emphasied that this operation will not relieve the oedema but will relieve venous stasis.

Deep appreciation must be paid to Dr. J. Prestwich, Medical Superintendent, Edenvale Hospital, for allowing me to utilize Edenvale Hospital records and also to the registrars, interns and nursing staff who have contributed to the excellent pre-operative and post-operative management of the cases referred to here. My sincere thanks to Dr. H. B. Stein who, as usual, has been most helpful with his criticism of this paper.

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THE RELATION BETWEEN THE AGES AND THE INTELLIGENCE OF PATIENTS AT THE ALEXANDRA INSTITUTION

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In a random sample of the normal European population we do not expect to find any significant relationship between the chronological ages of individuals and their IQs. Presumably the most intelligent 20% will have the same average age as the middle 60% or as the dullest 20%. Stated otherwise, the younger generation will have about the same average IQ as the older.

With mental defectives, however, we expect to find some relationship between age and IQ, because we know that idiots have a shorter span of life than morons. Tredgold[1] devotes a chapter to a general discussion of the problem, but no systematic statistical study of it has as yet been recorded, and as accurate information may be of value in research in hospital planning and policy, and as it may add to our knowledge in the field of expectation of life, as well as promote our knowledge of the mentally defective, this work was undertaken to obtain precise data.

The subjects for the investigation were 863 patients on the register of the Alexandra Institution for Mental Defectives, one of the 3 institutions for mental defectives administered by the Union Health Department.

The general method employed was to compute and then to compare with one another the average ages of all the patients falling within increasing step-intervals of IQ from zero upwards, to compare with one another the average IQs computed in respect of different age levels, and to find correlation coefficients.

The age and IQ data were taken as at 31 December 1954. The intelligence of the patients had previously been tested by the writer. Gesell's tables of development[2] were used to assess the lowest mental ages, the Merrill-Palmer scale[3] for mental ages of 18-48 months, the Terman-Merrill scale[4] for mental ages of 6-12 years, and the National Bureau test[5] for 3-12 years. Raven's Progressive Matrices[6] and an unpublished test were used

as a non-verbal scale for deaf-mutes with relatively high mental ages, and for other patients with language complications. The IQs can be regarded as reasonably reliable.

Although fractions of a year and of IQ were employed in the original computations, they are omitted in the text in order to make the comparisons more readable and to condense the tables.

Table I reflects the number of patients found in each step-interval of IQ, and their corresponding average

TABLE I. CHRONOLOGICAL AGES IN YEARS OF 863 PATIENTS OPPOSITE INCREASING IQ INTERVALS

IQ	*Number*	*%*	*Average age*	*Median age*	*Standard deviation*
(0—4)	(32	4	15	15	7·2)
0—9	95	11	18	16	9
10—19	103	12	23	20	13·2
20—29	159	18	29	28	15·4
30—39	117	14	32	29	16·5
40—49	146	17	37	37	15·3
50—59	111	13	34	34	14·9
60—69	93	11	35	34	13·3
70+	39	5	31	31	10·9
Total	863	101	30·3	28·2	15·4

chronological ages. There is a convincing relationship between age and IQ; they increase concomitantly up to an optimum IQ of 45 (the mid-point of the step-interval) after which the average falls to a level of 31 years, when an IQ of 70 and higher is reached.

Two major factors operate in this relationship. The higher the IQ, the bigger is the life span, and this positive relationship would have continued above the IQ of 45, if it were not for a limit imposed by a second factor which is a negative one, namely that patients with IQs of about 55 and higher, unlike those with the lower IQs, tend to leave the institution before they reach the higher chronological ages, their withdrawal systematically depressing the average ages of the higher IQ categories. This trend is strongest in our highest IQ group where the spread of the ages as measured by the standard deviation has in consequence shrunk to 10·9 years.

Table II shows the incidence for each chronological age-interval from zero upwards and the corresponding

TABLE II. AVERAGE IQS OF 863 PATIENTS OPPOSITE INCREASING CHRONOLOGICAL AGE INTERVALS

Age	*Number*	*%*	*Average IQ*
0—9 ..	62	7	23
10—19 ..	200	23	27
20—29 ..	202	23	39
30—39 ..	162	19	42
40—49 ..	138	16	45
50—59 ..	72	8	41
60+ ..	27	3	44
Total ..	863	99	36·7 (S.D.20·2)

average IQs. The same striking relationship as in the previous table is revealed, the average IQ of patients of 30 years and older being twice that of those who are under 10, and average age and IQ increase concomitantly up to an optimum of 45 years after which the increase in IQ ceases. The drop in the IQ at 55 years is a chance irregularity in the distribution.

The relation so far revealed can be expressed by a single quantity, the correlation coefficient. (If the relation between the 2 variables is perfect, so that the person with the highest IQ is also the oldest, while the one with the next highest IQ is also the next in age and so on for all 863 patients, downwards to the one with the lowest IQ who will then also be the youngest, then the correlation is positive and perfect, and we have a correlation coefficient (r) of 1·00; if there is no relationship between the two variables, then the result is r·00. Between these extremes any values of r can be found). The r for our data, calculated by the product-moment method was found to be ·306 which was much higher than anticipated.

In search of an explanation for such a high correlation we next considered the possibility of the existence of large clinical groups of patients with a very high r between age and IQ, and who are perhaps responsible for the original high relationship. If we could identify such clinical varieties, extract them from the main inmate body and then compute fresh correlation coefficients and tables showing the relationship between age and IQ in respect of the residual group, a lowered r could be expected.

Various clinical varieties were submitted to a preliminary trial, until we arrived at 3 varieties with high age-IQ relationships—the spastics, the epileptics, and the spastic epileptics, the last constituting an overlap of the first 2; they numbered 159, 163 and 62 respectively, making a total of 260 Ss.

Table III reflects the incidence for each IQ step-interval and the corresponding average age, the 3 clinical

TABLE III: AVERAGE AGES OPPOSITE INCREASING IQ-INTERVALS OF 159 SPASTICS, 163 EPILEPTICS, 62 SPASTIC EPILEPTICS AND THE THREE GROUPED

IQs	*Spastics*		*Epileptics*		*Spastic Epileptics*		*Grouped*	
	No.	*Age*	*No.*	*Age*	*No.*	*Age*	*No.*	*Age*
(0—4)	(26	13	18	13	17	13	27	13)
0—9	53	16	42	15	30	14	65	16
10—19	25	16	25	19	11	17	39	18
20—29	30	28	28	26	9	21	49	28
30—39	18	28	15	31	6	26	27	30
40—49	13	36	26	34	6	37	33	34
50—59	20	35	13	34	—	—	33	35
60+	—	—	14	33	—	—	14	33
Total	159		163		62		260	
Ave. age	23		25		19		25·8	
Ave. IQ	24		28		18		29·4	
r Age-IQ	·539		·527		·608		·515	

varieties being treated first separately and finally grouped.

The relationships are considerably higher than those found for the total inmate body (Table I). The average age of the highest IQ levels is 3 times that of the low grade idiots, and each increase in the IQ interval is accompanied by a corresponding increase in average age. These general findings are confirmed by the exceptionally high values for r.

These groups differ from the total inmate body in that there is no drop or levelling out of the average ages when the higher IQs are reached, and therefore, only one factor is required to account for the relationship revealed, namely that the higher the IQ, the bigger is the expecta-

tion of life. In consequence of their superimposed handicap the members of these groups do not leave the institution, so that the 2nd factor, earlier invoked, is absent here. The circumstance that for all 3 clinical varieties the final average age and IQ for each group are almost identical is a chance occurrence.

We next calculated in the same manner as before, the average IQs in respect of each clinical variety separately and finally grouped. The data are presented in Table IV.

TABLE IV. AVERAGE IQS OPPOSITE INCREASING AGE-INTERVALS OF 159 SPASTICS, 163 EPILEPTICS, 62 SPASTIC EPILEPTICS AND THE THREE GROUPED

Ages	*Spastics*		*Epileptics*		*Spastic Epileptics*		*Grouped*	
	No.	*IQ*	*No.*	*IQ*	*No.*	*IQ*	*No.*	*IQ*
0—9	29	14	16	11	15	9	30	15
10—19	50	21	57	19	24	9	83	23
20—29	35	28	40	33	14	29	61	31
30—39	25	35	23	36	9	30	39	37
40—49	12	35	16	46	—	—	28	41
50+	8	44	11	42	—	—	18	43
Total	159		163		62		260	

The same relationship as observed under Table III emerges and no special comment is called for—patients who reach the age of 35 have an average IQ 3 times that of children under 10, and the maximum age reached by spastic epileptics is only 39 as against 50+ years for the rest.

The 260 clinical cases were next extracted from the main body of patients and fresh data computed to show

TABLE V. AVERAGE AGES AT INCREASING IQ-INTERVALS OF 603 PATIENTS OPPOSITE EXTRACTED CLINICAL VARIETIES

IQ	*Clinical Groups*			*Residual Group*		
	No.	*% of 260*	*Age*	*No.*	*% of 603*	*Age*
(0—4)	(27	(10)	13	5	(1)	19)
0—9	65	(25)	16	30	(5)	22
10—19	39	(15)	18	65	(11)	26
20—29	49	(19)	28	110	(18)	30
30—39	27	(10)	30	90	(15)	33
40—49	33	(13)	34	108	(18)	37
50—59	33	(13)	35	87	(15)	35
60—69	14	(5)	33	77	(13)	35
70+	—	—	—	36	(6)	31
Total	260			603		
Ave. age	25·8 yrs.			32·3 yrs.		
r Age-IQ	·515			·184		

the age-IQ relationship for the residual of 603 Ss. The data for the 260 subjects and for the 603 are set out alongside each other in Table V, which reflects the average ages corresponding to increasing IQ intervals. The residual group are superior to the special clinical varieties; their average age is higher, relatively fewer of them are in the low-grade idiot category and they reach a higher maximum IQ, 4 times as many being in the IQ category of 60+.

The relationship between age and IQ persists but it has been greatly reduced, the age range of the residual being now 19-37 years as against 15-37 for the original 863 Patients (Table I). The reduced relationship is also reflected in the considerably lowered value of r, which is now ·184.

The average IQs of the residual group were computed and set out against those of the clinical groups but, as no fresh valuable information emerged, they are not tabulated here. The residual had a higher average IQ than the clinical, more of them reaching the highest age levels. The extraction of the clinical group had the effect of reducing the relationship between age and IQ, so that the new average IQ range is 30-45 as against 23-45 (Table II), but the restant relationship remains high.

Attempts at locating other clinical groups for exclusion from the main body in a statistically reliable manner, in order further to reduce our correlation coefficient were unsuccessful.

To facilitate comparisons, Table VI shows the final state of the average ages of the different groupings, their average IQs and the correlation coefficients.

TABLE VI. FINAL AGES, IQS AND CORRELATION COEFFICIENTS

Category	*No.*	*Age*	*IQ*	*r*
Spastic epileptics	62	18·7	18·2	·608
Spastics inclusive of spastics with epilepsy	159	23·4	23·6	·539
Epileptics inclusive of epileptics with spasticity ..	163	25·2	28·2	·527
Spastics without epilepsy ..	98	26·4	27·5	·441
Epileptics without spasticity	101	28·7	34·7	·382
Total patients	863	30·3	36·7	·306
Residual after excluding of epileptics	700	31·4	38·7	·239
Residual after excluding of epileptics and spastics ..	603	32·3	40·4	·184

It epitomizes much of the preceding data and calls for no special comment, its most striking feature being the inverse relationship between the correlation coefficients on the one hand, and age and IQ on the other. The hierarchy is almost perfectly regular.

It remains to present a percentage table (Table VI) of the distribution of IQs against an age scale. It has practical value, besides bringing out many of our earlier findings in full relief. Much of the data in Table I is

TABLE VII. PERCENTAGE PATIENTS UNDER THEIR RESPECTIVE IQ INTERVALS LOCATED OPPOSITE EACH AGE RANGE

Ages	*Intelligence Quotients*								
	(0 *to* 4)	0 *to* 9	10 *to* 19	20 *to* 29	30 *to* 39	40 *to* 49	50 *to* 59	60 *to* 69	70 *to* 79
0—9	25	17	14	10	6	1	4	2	3
10—19	53	53	38	22	21	15	14	9	10
20—29	22	18	22	22	26	19	25	28	36
30—39		9	14	20	15	22	22	25	28
40—49		3	8	13	18	21	21	24	23
50—59			4	10	8	16	11	8	
60—69			1	3	3	2	5	3	
70—79					3	2		1	
80—89						1			
No.	(32)	95	103	159	117	146	111	93	39
Ave. age	15	18	23	29	32	37	34	35	31
Med. age	15	16	20	28	29	37	34	34	31

represented in visible and readable form. It is noted that from IQ 40 upwards the distributions are nomal, the average and median ages being identical; below IQ 40 the median ages are smaller than the average ages, the distributions are negatively skewed by an accumulation of cases at their lower ends, The table lends prominence to the paucity of cases in the quadrant bounded by ages

50-89 and IQs 50-79, this being the result of the second factor identified under Table I.

DISCUSSION

Subscribing to the traditional assumption that the coefficient of correlation for chronological age and IQ is ·00 in a random sample of the population, we set out to investigate the position in institutionalized mental defectives, and found what must be a significant discovery, a positive relationship varying from r ·184 to r ·608, the value depending upon the clinical grouping. We cannot concern ourselves here with the theoretical implications of this finding, but its practical value for the management of mental defectives merits brief consideration.

1. The correlation coefficients reveal that, in terms of averages, patients with high IQs also tend to reach high chronological ages, but the discovery of a second factor which operates in the age-IQ relationships shows that high-grade patients do not remain indefinitely in the institution. Table VII shows that they leave mainly from the age of 35-45 years, an age at which it has become difficult for them to throw off attitudes and standards engendered in an institutional environment of mental defectives, to learn a set of new social and ethical standards.

The question may be posed whether it has been the best plan to keep them so long in an institution or even grant them admission in the first instance. The most suitable place for children with IQs 50+ who are unencumbered with superimposed handicaps are the Provincial Education Department's special classes and, failing these, the homes for backward children administered by the Union Education Department. Only when these bodies have cared for such children up to the age of 19 years, at which the provisions of the Children's Act of 1937 cease to apply, only then should they properly be considered for an institution for mental defectives when all else has failed.

Table VII shows that this observation concerns a fair number of patients, and our findings enable us to approach the problem of allocation of hospital accommodation more scientifically by helping them to anticipate and hasten their inevitable departure.

2. Children with IQs 0-4 are very deeply defective and their expectation of life is poor. Our findings (Tables I and III) show that there are 32 in this group, of whom 17 are spastic epileptics; and that in the IQ range of 0-9, there are 95 patients of whom 30 are spastic epileptics, 12 are epileptics only and 23 are spastics only. They—and one could include a fair proportion of the 103 patients in the IQ 10-19 range—seem to constitute a rather distinct clinical group, different from the other patients in their material hospital requirements. It would appear as if a bold, scientifically orientated policy would call for their complete segregation from other mental defectives, in an environment devised and standardized for their specific needs.

3. We now know that all infants have not the same statistical expectation of life. We have found correlation coefficients for age and IQ which range from ·184 to ·608, the value varying with the absence or presence of superimposed specific handicaps. This knowledge, together with the data in Table I, enables us to assess more precisely than hitherto an infant's expectation of life, when backwardness and the degree of it are diagnosed.

SUMMARY

This statistical study aims at making our knowledge of mental defectives more exact by evaluating the relation between their average age and IQ.

The ages of patients in different IQ categories were computed and compared; similarly, average IQs of different age-groups were found and compared. Correlation coefficients were found.

A percentage table showing the age limits within which given percentages of patients with known IQs fall, was compiled.

An unmistakeable relation between age and IQ was found, its extent depending upon the degree of defect and its complications.

The findings have academic and practical value.

Grateful acknowledgements are due to Dr. M. Cohen, the Physician Superintendent of The Alexandra Institution for his assistance, and to Professor I. R. Vermooten, the Commissioner for Mental Hygiene, for his encouragement and for permission to publish.

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UNION DEPARTMENT OF HEALTH BULLETIN

Union Department of Health Bulletin. Report for the 7 days ended 3 April 1956.

Plague, Smallpox, Typhus Fever: Nil.

Epidemic Diseases in Other Countries.

Plague: Nil.

Cholera in Calcutta (India); Chittagong, Dacca (Pakistan).

Smallpox in Kandahar (Afghanistan); Moulmein, Rangoon (Burma); Phnom-Penh (Cambodia); Ahmedabad, Bombay, Calcutta, Delhi, Madras, Pondicherry, Visakhapatnam (India); Chittagong, Dacca, Lahore (Pakistan); Saigon-Cholon, Tourane (Danang) (Viêt-Nam); Nairobi (Kenya).

Typhus Fever in Kabul (Afghanistan); Alexandria (Egypt).

Union Department of Health Bulletin: Report for the 7 days ended 12 April, 1956.

Plague, Smallpox, Typhus Fever: Nil.

Epidemic Diseases in other Countries.

Plague: Nil.

Smallpox in Moulmein, Rangoon (Burma); Phnom-Penh (Cambodia); Ahmedabad, Allahabad, Bombay, Calcutta, Delhi, Jodhpur, Madras, Visakhapatnam (India); Huê (Viet-Nam); Mombasa (Kenya).

Cholera in Calcutta (India).

Typhus Fever in Alexandria (Egypt).

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

MEDICAL AID SOCIETIES

The official list of recognized societies as at 1 May 1956 is published below for general information. This replaces the list in the present Tariff Book.

L. M. Marchand
Associate Secretary

Medical House
Cape Town
19 April 1956

MEDIESE HULPVERENIGINGS

Die offisiële lys van erkende verenigings, met ingang 1 Mei 1956, word vir algemene inligting hieronder gepubliseer. Dit vervang die lys in die huidige tarieweboek.

L. M. Marchand
Medesekretaris

Mediese Huis
Kaapstad
19 April 1956

APPROVED MEDICAL AID SOCIETIES : GOEDGEKEURDE MEDIESE HULPVERENIGINGS

1. A.A. Mutual Medical Aid Society, P.O. Box 9595, Johannesburg.
2. Abercom Group Sick Benefit Society, P.O. Box 715, Port Elizabeth, C.P.
3. African Cables Medical Benefit Fund, P.O. Box 172, Vereeniging, Transvaal.
4. African Explosives Medical Aid Society, P.O. Box 1122, Johannesburg.
5. African Homes Trust Sick Fund, 28 Wale Street, Cape Town.
6. African Oxygen & Acetylene Medical Aid Society, P.O. Box 5404, Johannesburg.
7. Afrikaanse Pers Beperk se Siektefonds, Posbus 845, Johannesburg.
8. Alex. Aiken & Carter Medical Benefit Society, P.O. Box 2636, Johannesburg.
9. Algoa Medical Aid Society, P.O. Box 369, Port Elizabeth, C.P.
10. Anglo-Alpha (Dudfield) Medical Aid Society, Private Bag, P.O. Lichtenburg, T'vaal.
11. Anglo-Alpha (Roodepoort) Benefit Society, P.O. Box 15, Roodepoort, Transvaal.
12. Argus Medical Benefit Society (Cape Argus Branch), St. George's Street, Cape Town.
13. Argus Medical Benefit Society (Daily News Branch), P.O. Box 1491, Durban.
14. Argus Medical Benefit Society (Star Branch), P.O. Box 1014, Johannesburg.
15. A.T.I. Medical Aid Society, P.O. Box 5057, Boksburg North.
16. Atlantic Refining Company Medical Aid Society, P.O. Box 664, Cape Town.
17. Babcock and Wilcox Medical Aid Fund, P.O. Box 545, Vereeniging.
18. Bakers Ltd. European Employees' Sick Benefit Fund, P.O. Box 692, Durban.
19. Bloemfontein Municipal Employees' Medical Aid Society, P.O. Box 288, Bloemfontein.
20. Boksburg Municipal Employees' Medical Aid Fund, P.O. Box 215, Boksburg.
21. Broderick Medical Aid Society, P.O. Box 186, Vereeniging.
22. Building Societies Joint Medical Aid Fund, P.O. Box 5728, Johannesburg.
23. S. Butcher & Sons Ltd. Medical Aid Society, P.O. Box 1004, Durban.
24. Caltex Medical Aid Society (S.A.), P.O. Box 714, Cape Town.
25. Cape Times Medical Aid Society, P.O. Box 11, Cape Town.
26. Cape Town Municipal Employees' Medical Aid Society, P.O. Box 1939, Cape Town.
27. Central News Agency Limited Medical Benefit Society, P.O. Box 1033, Johannesburg (excluding Cape Town and suburbs, Durban municipal area, Johannesburg and Witwatersrand, and Port Elizabeth and Pretoria municipal areas).
28. Civil Service Medical Benefit Association, P.O. Box 176, Pretoria.
29. Consolidated Glassworks Limited Medical Aid & Sick Benefit Society, P.O. Box 562, Germiston.
30. Corner House Insurance Fund, P.O. Box 1056, Johannesburg.
31. Coronation Medical Aid Society, P.O. Box 1517, Durban.
32. Crookes Bros. Limited Medical Benefit Fund, 301 Smith Street, Durban.
33. D.F.A. Medical Benefit Society, P.O. Box 610, Kimberley.
34. Eastern Province Cement Co. Ltd. Medical Aid Society, P.O. Box 2016, Port Elizabeth.
35. E.P. Newspapers Medical Aid Society, P.O. Box 1117, Port Elizabeth.
36. E.D.C. Industries Medical Aid Society, P.O. Box 3448, Johannesburg.
37. Egnep Medical Aid Society, P.O. Penge, Transvaal.
38. Elwamba Medical Aid Fund, c/o E.L. Wool Exchange Limited, 50/52 Church Street, East London.
39. Escom (Natal Central Undertaking) Medical Benefit Society, P.O. Box 30, Colenso, Natal.
40. Everite Medical Aid Society, P.O. Kliprivier, Transvaal.
41. Federated Employers' Medical Aid Society, P.O. Box 666, Johannesburg.
42. Federation of Master Printers of S.A. Medical Aid Society, P.O. Box 1200, Johannesburg.
43. Ford Medical Aid Society, P.O. Box 788, Port Elizabeth.
44. Friend Medical Aid Fund, P.O. Box 245, Bloemfontein.
45. General Motors Medical Aid Scheme, P.O. Box 1137, Port Elizabeth.
46. Germiston Industries Medical Aid Society, 113 Pylon House, Herman Street, Germiston.
47. Gledhow-Chaka's Kraal Sugar Co. Ltd. Medical Benefits Fund, 301 Smith Street, Durban.
48. Globe Medical Aid Society, Railway Street, Woodstock, Cape Town.
49. Greaterman's Pension Benefit and Welfare Society (all Branches), P.O. Box 5460, Johannesburg.
50. Hollerith Medical Aid Society, P.O. Box 7018, Johannesburg.
51. Hubert Davis Johannesburg Staff Medical Aid Society, P.O. Box 1386, Johannesburg.
52. Sir J. L. Hulett & Sons Ltd. Medical Benefit Fund, P.O. Box 248, Durban.
53. Hume Cape Medical Benefit Society, P.O. Box 7, Bellville, C. P.
54. Hume (Transvaal) Medical Benefit Society, P.O. Box 204, Germiston.
55. Hunt, Leuchars & Hepburn Ltd. (Durban) Employees' Medical Benefit Fund, P.O. Box 943, Durban.
56. Hunt, Leuchars & Hepburn Ltd. (Transvaal Staff) Medical Aid Society, P.O. Box 47, Johannesburg.
57. Iscor Medical Benefit Fund, P.O. Box 450, Pretoria.
58. I.W.S. Medical Aid Society, P.O. Box 6946, Johannesburg.
59. J. W. Jagger & Co. Ltd. Medical Aid Society, P.O. Box 726, Cape Town.
60. Johannesburg Board of Executors' Medical Aid Society, P.O. Box 271, Johannesburg.
61. Joseph Liddle (Pty.) Ltd. Medical Aid Society, P.O. Box 106, Johannesburg.
62. Klerksdorp Munisipale Werknemers Siektefonds, Posbus 99, Klerksdorp.
63. K. & L. Timbers Ltd., P.O. Box 6994, Johannesburg.
64. Koegas Medical Aid Society, P.O. Koegasbridge, C.P.
65. Krantzberg Mines Medical Aid Society, P.O. Box 18, Omaruru, S.W.A.
66. Kroonstad Munisipale Mediese Hulpvereniging, Posbus 302, Kroonstad.
67. G. H. Langler & Co. Medical Aid Society, P.O. Box 3762, Johannesburg.
68. Legal & General Medical Aid Scheme, P.O. Box 4870, Johannesburg.

69. Mail, Times & Express Medical Aid Society, c/o Rand Daily Mail, Ltd., P.O. Box 1138, Johannesburg.
70. L. H. Marthinusen Medical Aid Society, P.O. Box 64, Denver, Johannesburg.
71. Masonite Medical Aid Society, P.O. Box 57, Estcourt, Natal.
72. Matabeleland Medical Aid Society, P.O. Box 1776, Bulawayo, Southern Rhodesia.
73. Metal Box Company of S.A. Medical Aid Society, P.O. Box 7752, Johannesburg.
74. Municipal Employees' Medical Aid Society (Durban), P.O. Box 625, Durban.
75. Natal Building Society Medical Aid Fund, P.O. Box 947, Durban.
76. Natal Coal Owners' (Durban Staff) Medical Aid Society, P.O. Box 281, Durban.
77. Natal Estates Sick Fund Benefit Society, P.O. Mount Edgecombe, Natal.
78. Natal Industries Medical Aid Society, P.O. Box 1300, Durban.
79. N.T.E. Staff Medical Aid Fund, P.O. Box 39, Pietermaritzburg.
80. National Industrial Credit Corporation Medical Aid Society, P.O. Box 8296, Johannesburg.
81. National Portland Medical Aid Society, P.O. Box 21, Claremont, C.P.
82. New Consolidated Goldfields Employees' Medical Aid Fund, P.O. Box 1167, Johannesburg.
83. Northern Medical Aid Society, P.O. Box 3437, Johannesburg.
84. Northern Rhodesia Civil Servants Medical Aid Society, P.O. Box 294, Lusaka, Northern Rhodesia.
85. Norwich Union Life Insurance Society Staff Medical and Surgical Benefit Scheme, P.O. Box 1226, Cape Town.
86. Ore & Metal Medical Aid Society, P.O. Box 3548, Johannesburg.
87. Pietermaritzburg Chamber of Industries Medical Aid Society, P.O. Box 365, Pietermaritzburg.
88. Polliack Group Medical Aid Society, P.O. Box 3008, Johannesburg.
89. Pongola Sugar Milling Co. Ltd. Medical Benefit Fund, P.O. Box 194, Durban.
90. Post Office Medical Aid Society, P.O. Box 303, Germiston.
91. Pretoria Municipal Employees' Sick Fund, P.O. Box 408, Pretoria.
92. Pretoria News Medical Benefit Society, P.O. Box 439, Pretoria.
93. Pretoria Portland Cement Co. Ltd. No. 1 Works (Hercules) Medical Aid Society, P.O. Box 405, Pretoria.
94. Pretoria Portland Cement Co. Ltd. No. 2 Works Medical Society, P.O. Box 7, Slurry, Western Transvaal.
95. Pretoria Portland Cement Co. Ltd. No. 3 Works (Jupiter) Medical Aid Society, P.O. Box 73, Cleveland, Transvaal.
96. Pretoria Portland Cement Co. Ltd. No. 4 Works Medical Aid Society, P.O. Box 26, Orkney, district Klerksdorp.
97. Printing Industry Medical Aid Society, P.O. Box 1993, Pretoria.
98. Rand Public Service Medical Aid Society, P.O. Box 28, Boksburg.
99. Rand Water Board Sick Fund, P.O. Box 1127, Johannesburg.
100. Randle Bros. & Hudson Ltd. (Durban) Sick Benefit Fund, P.O. Box 1046, Durban.
101. Randles Bros. & Hudson Ltd. (Johannesburg) Employees' Sick Benefit Fund, P.O. Box 2678, Johannesburg.
102. Reckitt & Colman Medical Aid Society (S.A.), P.O. Box 1097, Cape Town.
103. 'Rennie' & 'The Consolidated' Employees' Medical Aid Fund, P.O. Box 1006, Durban.
104. Reunert & Lenz Ltd. Medical Aid Society (All Branches), P.O. Box 92, Johannesburg.
105. Reynolds Bros. Ltd. Medical Benefits Fund, 301 Smith Street, Durban.
106. E.S. & A. Robinson (Pty.) Ltd. Medical Aid Society, P.O. Box 293, Germiston.
107. Royal-Globe Medical Aid Fund, P.O. Box 317, Cape Town.
108. Safim Medical Aid Society, P.O. Box 233, Vereeniging.
109. Safmarine Medical Aid Society, P.O. Box 2171, Cape Town.
110. Safnit Mills Medical Aid Fund, P.O. Box 11, Jeppestown, Johannesburg.
111. Santam-Sanlam Siektefonds (Alle Takke), Posbus 1, Sanlamhof, K.P.
112. Sasol Medical Aid Society, P.O. Box 1, Sasolburg, O.F.S.
113. Schwartz, Fine, Kane & Co. Medical Aid Society, P.O. Box 5069, Johannesburg.
114. Shell Medical Aid Society (S.A.), P.O. Box 2231, Cape Town.
115. Siektefonds van Wolgroeiers Afslaers Beperk, Posbus 765, Port Elizabeth.
116. C. G. Smith & Co. Ltd. Medical Aid Fund, 301 Smith Street, Durban.
117. S.A. Association of Municipal Employees' (S.A.A.M.E.) Medical Aid Fund, P.O. Box 62, Pretoria.
118. S.A. Breweries Medical Aid Society, P.O. Box 1099, Johannesburg.
119. S.A.K.A.V. Sick Benefit Fund, P.O. Box 33, Paarl.
120. S.A. Mutual Fire & General Insurance Co. Ltd. Staff Medical Aid Fund, P.O. Box 516, Johannesburg.
121. S.A. Mutual Life Assurance Society Staff Medical Aid Fund, P.O. Box 66, Cape Town.
122. S.A. Press Association Medical Aid Society, P.O. Box 7766, Johannesburg.
123. S.A. Teachers' Association Medical Aid Society, 12 Bellevue Road, Sea Point, C.P.
124. S.A. Torbanite (Boksburg) Medical Aid Society, P.O. Box 83, Boksburg North.
125. Southern Medical Aid Society, P.O. Box 42, Cape Town.
126. Springbok Medical Aid Society, P.O. Box 7614, Johannesburg.
127. Standard Brass Medical Aid Society, P.O. Box 229, Benoni.
128. Stewarts & Lloyds Medical Benefit Fund, P.O. Box 74, Vereeniging.
129. Stuttafords Medical Aid Society, P.O. Box 69, Cape Town.
130. Sun Insurance Office Ltd. Staff Medical Aid Fund, P.O. Box 429, Johannesburg.
131. Traduna Medical Aid Fund, P.O. Box 8791, Johannesburg.
132. Transvaal Chamber of Mines Medical Aid Society, P.O. Box 809, Johannesburg.
133. Transvaal Corundum Associated Asbestos Medical Aid Society, P.O. Box 72, Pietersburg, Transvaal.
134. Transvaal Society of Accountants Medical Aid Society, P.O. Box 2995, Johannesburg.
135. Umzimkulu Sugar Co. Ltd. Medical Aid Fund, P.O. Box 43, Durban.
136. Union Flour Mills Sick Fund, P.O. Box 1010, Durban.
137. United Banks' Medical Aid Society, P.O. Box 1242, Cape Town.
138. United Building Society Medical Benefit Fund, P.O. Box 7735, Johannesburg.
139. University of the Witwatersrand (Johannesburg) Staff Medical Aid Fund, Milner Park, Johannesburg.
140. Vacuum Medical Aid Society, P.O. Box 35, Cape Town.
141. Village Board of Management of Welkom Medical Aid Society, P.O. Box 708, Welkom, O.F.S.
142. Wright Boag & Head Wrightson Sick Benefit Fund, P.O. Box 183, Benoni.
143. Yorkshire Medical Aid Society, P.O. Box 2755, Johannesburg.

MEDICAL BENEFIT SOCIETIES WHICH ALLOW FREE CHOICE OF DOCTOR FOR SPECIALIST SERVICES ONLY: MEDIESE BYSTANDSVERENIGINGS WAT VRY KEUSE VAN DOKTER ALLEEN VIR SPESIALISTEDIENSTE TOELAAT

1. Begbie Medical Benefit Fund, P.O. Box 192, Middelburg, Transvaal.
2. Breyten Coalfields Benefit Society, P.O. Box 110, Ermelo.
3. Broken Hill Mine Employees' Medical Specialist Fund, P.O. Box 45, Broken Hill.
4. De Beers Consolidated Mines Limited Benefit Society, P.O. Box 616, Kimberley.
5. Durban Roodepoort Deep Limited Benefit Society, P.O. Box 193, Roodepoort.

6. Jagersfontein Mine Benefit Society, P.O. Box 2, Jagersfontein, O.F.S.
7. Krugersdorp Municipal Employees' Medical Benefit Society, P.O. Box 101, Krugersdorp.
8. Northern Rhodesia Mine Employees' Medical Fund, P.O. Box 134, Kitwe, Northern Rhodesia.
9. Public Utility Transport Corporation Staff Sick Fund, P.O. Box 9571, Johannesburg.
10. Randfontein Estates Employees' Sick Benefit Society, P.O. Box 37, Randfontein.
11. Roodepoort-Maraisburg Municipal Employees'. Association Sick Benefit Society, P.O. Box 217, Roodepoort.
12. Roodepoort-Maraisburg Non-Scheduled Mines' and Industries' Benefit Society, P.O. Box 225, Roodepoort.
13. Rosherville-Maraisburg Benefit Society, P.O. Box 99, Cleveland, Johannesburg.
14. Simmer Pan Medical Benefit Society, P.O. Box 103, Germiston.
15. Springs Mines Benefit Society, P.O. Box 54, Springs.
16. Springs Municipal Employees' Association, P.O. Box 45, Springs.
17. Transvaal Jewellers' and Goldsmiths' Sick Benefit Fund, P.O. Box 8530, Johannesburg.
18. Vereeniging Power Station Medical Society, P.O. Box 27, Vereeniging.
19. Witbank Coalfields Benefit Society, P.O. Box 26, Witbank.
20. Witbank Power Station Medical Aid Society, P.O. Box 197, Witbank.

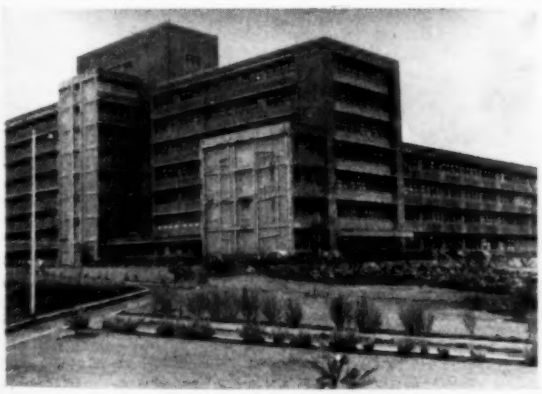
Note (1) The Federal Council of the Medical Association of S.A. has extended the operation of the Tariff to the personnel and dependants, pensioners and their dependants of the High Commission Territories (Basutoland, Bechuanaland Protectorate and Swaziland). Persons entitled to such concessions will be supplied with letters of introduction signed by the Secretaries of the Territories concerned.

Note (2) The Tariff also applies to additional medical assistance rendered to members of the Police Force, Prison Officers, members of the Permanent Defence Forces and the wives and unmarried children under the age of 18 years of European personnel of these Departments. In addition it applies to all persons for whose medical attention the Government is responsible in the Departments of Education, Arts and Science; Health; Pensions and Social Welfare. Such fees will be subject to a reduction of 10% on services to a patient costing up to 25 guineas and of 15% on those costing over 25 guineas, but no reduction will apply to any account of £2 2s. 0d. or less in respect of services rendered to any one patient.

EDENDALE HOSPITAL, PIETERMARITZBURG, NATAL

The building of this hospital represents one of the most important recent steps taken by Natal to provide for the steady increase in the numbers of Bantu and Indian sick.

In December 1950 the Administrator of Natal appointed a commission of inquiry to investigate the shortage of hospital beds in Natal. The Commission's report (No. NP 6/1952), a model of its kind, among other recommendations noted and endorsed the plans formulated by the Natal Administration for the sick of the Midlands: these plans included the building of a large non-European hospital at Edendale, 5 miles from Pietermaritzburg,



A view of Edendale Hospital.

to serve as the Midlands Regional Hospital with satellite hospitals at Polela, Estcourt, Greytown etc. Hitherto Grey's Hospital, Pietermaritzburg, had been the only large hospital in the Midlands of Natal, but accommodation there was inadequate, siting was poor as regards the non-European needs, there was no room for expansion, and there were no facilites for the training of Indian or Bantu nurses.

The main reasons for the shortage of beds was a rapid increase in the Indian population and an increasing hospital-consciousness on the part of the Native population.

The highly-populated Native area of Edendale was deemed the most suitable site for the new hospital, the needs of the town non-Europeans being met by the planning of a large reception centre and out-patient department in the Native quarter of Pietermaritzburg.

The erection of the building was begun in 1951, planned as a multi-storied cross of Lorraine and initially designed to accommodate 620 beds but allowing optional expansion to double that number should the need arise later. Ancillary buildings include a large Bantu nurses' home and a training centre both for general and midwifery training.

The hospital was opened for patients early in 1954.

CLINICAL DEPARTMENTS

Medical Division

A full-time specialist is in charge of this department and is assisted by 3 part-time visiting physicians. Other staff include 2 medical registrars and 4 house physicians.

Excluding the paediatrics section (see below), the medical beds number 150 equally divided between male and female, housed in 4 wards. All essential equipment such as the electrocardiograph is new and satisfactory. A small laboratory is available for the staff of the medical division.

The work of the Division is varied in quality and more than sufficient in quantity, with a monthly turnover of 300 patients. The diseases seen are those both of the tropics and of the temperate zones, with emphasis on malnutrition, bowel infections, phthisis, and heart diseases.

House physicians normally spend 6 months with the medical division, which includes a period of work in the paediatrics wards.

Surgical Division

This department is in charge of a full-time specialist surgeon with a visiting staff of 2 general surgeons (one specialist) and one surgeon specialist each for the sub-departments of ophthalmology, oto-rhino-laryngology, oro-facio-maxillary surgery, and urology. The department of orthopaedics (see below) is under the care of a visiting surgeon. The junior staff consists of 5 registrars and 5 house surgeons.

General surgical beds number approximately 100, those for orthopaedics 130 and those for sub-departments approximately 15 each.

An important section of the department is a special resuscitation ward, adjacent to the theatre block, in which are centralized all gravely ill patients of whatever sort. Each bed has piped oxygen and suction, and blood is kept readily available in the ward for transfusion.

The turn-over of general surgical patients numbers about 150 monthly, covering a wide variety of conditions.

Department of Obstetrics and Gynaecology

The obstetrics section of 100 beds occupies the whole of the third floor of the building, and the gynaecology section has a further 34 beds. A full-time specialist is in charge, assisted by a part-time specialist. Other staff include 2 registrars and 3 house-surgeons.

Hospital deliveries number about 300 patients monthly, excluding those delivered by the district maternity workers controlled from the hospital. All abnormalities are seen, the incidence of operative deliveries now averaging about 10% of the total deliveries. Antenatal clinics are held 4 times a week and are well-attended, with a monthly average of about 900 cases, including repeat visits. No postnatal clinics are held but selected cases are referred to the gynaecology out-patient clinics for follow-up investigation.

In the gynaecology section 3 out-patient clinics and 3 operative sessions are conducted each week; the turn-over is satisfactory and the clinical material available provides excellent opportunities for the training of registrars and house surgeons, particularly because of the high incidence of contracted pelvis in the Bantu, a problem at present under investigation.

Pediatric Section

This occupies the whole of the top floor of the hospital and, as stated above, cares for about 100 children between the ages of 1 and 9 years. It comprises a variety of small wards, a reception ward, crib room, solarium etc.

The work is in charge of a part-time visiting paediatrics specialist assisted by a registrar and 2 house physicians. During the first year of working 1,200 patients were treated in this department, many of them suffering from malnutrition or tuberculosis—diseases especially common among illegitimate children.

Orthopaedics Section

The orthopaedics section as stated above, has at its disposal about 130 beds, and a staff of one visiting orthopaedic surgeon, 2 registrars, and 2 house surgeons. Much bone and joint tuberculosis is seen in this hospital.

Other Sections

As stated above, other special sections include an eye section, an ear nose and throat section, and a facio-maxillary section, each with 15 beds and each under the care of a part-time visiting specialist. The hospital also houses a physiotherapy and rehabilitation department with a visiting specialist in physical medicine, and a dental department under the charge of a dental surgeon.

Out-patient Departments

There are 2 out-patient departments under the hospital control, one at Edendale Hospital itself, which handles about 10,000 attendances a month, while in Pietermaritzburg there is the town satellite centre, which is equipped with beds and an X-ray plant and handles about 12,000 patients a month.

ANCILLARY DEPARTMENTS

X-Ray Department

The department at Edendale Hospital comprises diagnostic and therapeutic sections, both of which embody the latest ideas in lay-out. The equipment is new and completely modern. Two major X-ray units are available, each capable of conventional and of high-kilovoltage technique; these are supplemented by a variety of smaller X-ray sets, each located in separate rooms.

The radiotherapeutics department is equipped with a fine, completely modern, automatic deep X-ray therapy set and a medium therapy set, both of which are extensively used.

The departmental staff include a full-time specialist, a head radiographer, and 6 junior radiographers.

Over 17,000 cases were seen in the last year; the large number of special investigations undertaken provides unique experience for radiographers interested in this type of work.

The radiologist at Edendale is also responsible for overseeing the work of the X-ray Department in the town satellite centre mentioned above.

Pathology Department

The laboratary is staffed by members of the Provincial Pathology Service, this staff consisting of 2 pathologists and 8 technicians; increase in staff is anticipated.

This laboratory does not suffer from the over-specialization now so serious a feature of large laboratories in general. It houses sub-departments of parasitology, bacteriology, serology, haematology, and morbid anatomy, and in addition is responsible for the running of a blood bank with a monthly consumption of 200 pints of blood. Very shortly the laboratory will also become responsible for the organization and staffing of a central sterilizing and syringe service.

In the current year the total examinations carried out numbered over 50,000. Approximately 40% of all hospital deaths reach the morbid anatomy department and the post-mortem examinations for 1955 numbered 550.

Anaesthetics Department

The staff consists of a full-time specialist and 3 registrars. The department is fully equipped with the most modern type of anaesthetic apparatus. Over 400 anaesthetics are administered monthly, of all types, and since a relatively high proportion of surgical patients arrive at the hospital *in extremis* this provides excellent training for the anaesthetics registrars and house staff.

The innovation of recovery wards has proved of great value; no patient leaves the recovery wards until the anaesthetist is satisfied he is fit to do so.

DISCUSSION

The opening of this large general hospital is of importance in that it provides excellent teaching facilities and a wide general experience for the junior medical officer, in which connection it is of interest to note that the hospital has been recognised by the South African Medical and Dental Council as a training centre for specialists in almost all branches of medicine and surgery. Similar recognition is being sought at the moment from the British Royal Colleges.

THE SOUTH AFRICAN PAEDIATRIC ASSOCIATION

The Second Congress of the South African Paediatric Association was held at Groote Schuur Hospital, Cape Town, from 19 to 21 January 1956. The Congress was attended by members and associate members from Cape Town, Pretoria, Johannesburg and Durban.

In his opening remarks, the President, Dr. H. L. Wallace, welcomed delegates and stressed the benefits, both academic and social, to be derived from a Congress such as this, in that it afforded an opportunity for paediatricians to make personal contact with their colleagues from the various centres throughout the Union. He also thanked the University authorities and members of the Cape Town Paediatric Sub-group for their help and cooperation in making the local arrangements for the holding of Congress.

In addition to the delegates, many of the meetings were attended by local practitioners and representatives of the Public Health and School Medical Services.

The papers presented by members covered a wide variety of topics and were as follows:

'Idiopathic Cardiomegaly', Dr. P. Smythe.

'Assessment of Degree of Sensitization in Haemolytic Disease of the Newborn (Rhesus) with treatment modified accordingly', Professor J. Davel.

'Reading Disability in Children', Dr. J. Rabkin.

'Neonatal Surgical Emergencies', Professor J. Louw.

'Coxsacki Myocarditis in the Newborn', Dr. S. Javett.

'The EEG in Childhood Convulsions', Dr. F. Friedlander.

In addition to the above, two general discussions were held, one on Kwashiorkor, in which Drs. Hansen, Walt, Suckling, Janssen

and Professor Davel reported on various investigations which they had carried out in connection with the disease. Rheumatic Fever in Childhood was the subject of the second discussion, which was opened by Dr. L. Braudo, who revealed that there had been a striking rise in the incidence of the disease in Johannesburg. Dr. Wallace, and Professors Ford and Davel then discussed the incidence of Rheumatic Fever in Durban, Cape Town and Pretoria and showed that, in these three centres, the disease was not apparently on the increase.

A part of one morning was devoted to an interesting clinical demonstration at Groote Schuur Hospital in which the following cases were shown:

'Vascular Ring after operation' and
'Absence of Peripheral Pulses', Professors Ford and Goetz.
'Ovarian Agenesis', Dr. Jackson.
'Fibrocystic Disease of the Pancreas', Dr. Hansen.
'Subclavian Aneurysm' and
'Tomato Baby', Dr. Smythe.
'Unusual Haemorrhages in the Newborn', Dr. Zilberg.

On the last morning, the Special Congress Lecture was delivered by Professor M. Van den Ende, who chose as his subject 'Active Immunisation Against Virus Diseases in Man'. This was received with acclamation by a large audience and the President warmly thanked Professor Van den Ende on behalf of the Association for his most interesting and stimulating address.

An interesting sidelight of Congress was a visit to the new Red Cross Memorial Children's Hospital at Rondebosch, when delegates were duly impressed by its magnificence.

Social functions included a very pleasant cocktail party at the home of Professor F. Ford, and a dinner followed by dancing held at the Palace Hotel, Claremont. The dinner was attended by Congress members and their wives and a number of distinguished guests were present. The toast of the South African Paediatric Association was proposed by Professor Ford, to which the President replied.

The Annual General Meeting of the Association was held during Congress and the following office-bearers were elected for the ensuing year. *President*—Dr. H. L. Wallace; *Secretary/treasurer*—Dr. F. Walt; *Executive Committee*—Dr. C. Comay; Professor J. Davel, Professor F. Ford, Dr. S. Heymann, and Dr. Pauline Klenerman.

PASSING EVENTS : IN DIE VERBYGAAN

Lede word daaraan herinner dat hulle die Sekretaris van die Mediese Vereniging van Suid-Afrika, Posbus 643, Kaapstad, sowel as die Registrateur van die Suid-Afrikaanse Mediese en Tandheelkundige Raad, Posbus 205, Pretoria, moet verwittig van enige adresverandering.

Versuim hiervan beteken dat die *Tydskrif* nie afgelewer kan word nie. Dit het betrekking op lede wat oorsee gaan sowel as dié wat binne die Unie van adres verander.

* * *

Dr. G. Reginald Crawshaw is no longer in partnership with Mr. L. Fatti, but is continuing his consultant practice as a thoracic surgeon at the Princess Nursing Home, Esselen Street, Johannesburg (Telephone: Rooms 44–2893, Residence 48–8488).

* * *

A Refresher Course for General Practitioners will be held by the University of Cape Town from Monday to Friday 23-27 July 1956. The course will include lectures, ward rounds, and demonstrations in Medicine, Surgery, Obstetrics and Gynaecology, and Specialities, which will be held at appointed times between 8 a.m. and 5 p.m. daily with two evening sessions at 8.15 p.m.

The number of practitioners accepted for the course will be restricted. A fee of £5 5s. is payable in advance to the Registrar, University of Cape Town, but not payable until after acceptance for the course. Board and lodging is available at the Medical Students' Residence for those desiring it, and for their wives, at the charge of £1 1s. per day per person.

Applications, stating whether residential accommodation will be required or not, should be submitted to the Registrar, University of Cape Town, Private Bag, Rondebsoch, by Saturday 2 June 1956.

Dr. Sam Galloon, F.F.A.R.C.S., D.A., formerly in private practice as an anaesthetist in Durban, has been appointed to the Lectureship in Anaesthetics at the University of Glasgow. Before coming to Durban Dr. Galloon had spent 3 years as Registrar and Senior Registrar to Professor W. W. Mushin at the Welsh National School of Medicine, Cardiff.

* * *

The Annual Dinner of the Cape Western Branch will be held on Friday, 15 June 1956, at Arthur's Seat Hotel, Beach Road, Sea Point. Tickets are obtainable at the office of the Branch, medical House, 35 Wale Street, Cape Town (P.O. Box 643), at a cost of 25s. each. All doctors visiting the Peninsula at that time are welcome.

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Erratum. 'Some Obstetrical Problems encountered in General practice' by A. Rosin, M.B., Ch.B. which appeared in the *Journal* of 28 April 1956 (page 407) was a paper presented at an ordinary general meeting of the Queenstown Division on 23 February 1956, and not, as stated, a paper presented at the South African Medical Congress, Pretoria, October 1955.

* * *

Dr. James Marshall, M.D., Dermatologist, formerly of Johannesburg and Pretoria, is now in practice at 610 Netherlands Bank Buildings, St. Georges Street, Cape Town. Telephones: Rooms 3–1461 (not 3–1416 as previously stated in error). Residence 8–4054.

REVIEWS OF BOOK : BOEKRESENSIES

BANTU GYNAECOLOGY

Bantu Gynaecology: By G. P. Charlewood, M.B., Ch.B., F.R.C.S. (Ed.), F.R.C.O.G. with contributions by O. S. Heyns, M.A., D.Sc., F.R.C.O.G. Pp. 172. 36s. Johannesburg: Witwatersrand University Press. 1956.

Contents: I. Some Ethnic Relations of the Bantu in South Africa, by O.S. Heyns. II. Congenital Malformations. III. Gonorrhoea. IV. Tuberculosis of the Female Genital Tract. V. Gynaecological Schistosomiasis. VI. Lymphopathia Venereum. VII. Abortion. VIII. Ectopic Pregnancy. IX. Advanced Extrauterine Pregnancy. X. Obstetrical Pressure Injuries. XI. Genital Prolapse, by O. S. Heyns. XII. Hydatidiform (Vesicular) Mole. XIII. Chorionepithelioma. XIV. Anomalous Genital Bleeding. XV. Fibromyomata. XVI. Carcinoma of the Vulva. XVII. Carcinoma of the Cervix. XVIII. Carcinoma of the Uterine Body. XIX. Ovarian Tumours. XX. Masculinisation of the Female. Bibliography. Index.

Having read Dr. Charlewood's book from cover to cover with very great pleasure and interest, I submit my impressions of the work as follows:

It is most fitting and proper that in a book describing the gynaecological peculiarities of the Bantu, the historical background of these peoples should be supplied in the first chapter. This erudite account makes unusually interesting and smooth reading and will be welcomed by both the overseas reader and our own South African medical man. Without doubt it creates the correct setting and atmosphere in which to unfold the remainder of the book.

Emphasis is naturally made on the differences between Bantu and European gynaecology. Congenital malformations are mentioned briefly, particularly the impact these have on the domestic and social aspect of Bantu life. It is interesting to note that pseudo-hermaphrodites are more commonly found in the Bantu than in the European in South Africa.

Gonorrhoea is one of the highlights of Bantu gynaecology and is fully dealt with. Gynaecological tuberculosis also is not uncommon among the Bantu; its effects on the vulva, vagina, cervix, uterus and adnexae are well described and treatment is briefly discussed. The gynaecological forms of bilharzia disease are also seen much more frequently in the Bantu female than

in the European. The precancerous nature of this infection is stressed, particularly when the cervix is involved. Lymphopathia venereum, a venereal disease seen almost exclusively among the Bantu population, is described fully.

Ectopic pregnancy forms a most important subject as it occurs so frequently among the Bantu and Coloured populations—accounting for nearly 6% of all gynaecological admissions. This follows on the high incidence of gonococcal salpingitis, the antibiotic treatment of which probably increases the chances of tubal pregnancy—an interesting point well brought out. This section rightly emphasizes the 'atypical' ectopic pregnancy which is met with almost as frequently as the classical picture of ectopic pregnancy. An excellent and most authoritative review of the subject is presented.

Vesico-vaginal fistula is a lesion that is unfortunately common among the Bantu female population. The etiology and treatment are clearly and concisely described by Dr. Charlewood, who, because of his wide experience in this field, must be considered an authority on the subject. One's only regret, however, is that this chapter is so short.

An outstanding feature of Bantu gynaecology is the rarity of genital prolapse, which is about 80 times more frequently met with in the European population of South Africa. This subject is expertly dealt with by Professor Heyns who advances a logical and original explanation for its rarity in the Bantu. In the same chapter Professor Heyns puts forward an original and thought-provoking explanation why the Manchester operation for prolapse is so successful. Metropathia haemorrhagica and endometriosis, which are also rare in the Bantu are discussed briefly.

Fibromyomata are found almost 3 times as commonly in the Bantu as in the European, and account for a considerable proportion of Bantu gynaecology. The ever-present demand by the Bantu patient to conserve her uterus has to be respected when treatment is considered.

Carcinoma of the genital tract is considered briefly and concisely. Carcinoma of the cervix is generally met with at an earlier age in the Bantu and its clinical diagnosis presents more problems than is usual in the European case. Carcinoma of the uterine body in the Bantu is very much less frequently seen than in the European. Similarly, ovarian neoplasms appear to occur less commonly in the Bantu, the only exception being dermoid tumours.

The photographs in this book are exceptionally good and are of a high standard. The line drawings are clear, simple and easy to follow. The print is clear, but wider spacing and larger margins would be an improvement.

The peculiarities of the Bantu female present a large and very fascinating mass of information to all who are interested in gynaecology. All this information is given clearly and concisely in a manner that is easily assimilable. The subjects raised, however, are often so interesting that one would have appreciated more detail and discussion. This book is unique in that it presents, for the first time, in a complete and well organized volume, the truly major differences between European gynaecology, as is found in all the standard text-books, and Bantu gynaecology as is seen in South Africa.

Dr. Charlewood, who is well qualified to have written such a book, is to be warmly congratulated on his achievement.

The book is an essential for all medical students in South Africa and it should be read by all who practice, or are likely to practice gynaecology among the Bantu peoples.

L. v. D.

PROLAPSE

Genital Prolapse and Allied Conditions. By Percy Malpas, Ch.M. (Liverpool), F.R.C.S. (Eng.), F.R.C.O.G. Pp. 199 + vii with 37 illustrations. 47s. 6d. London: Harvey & Blythe, Ltd. 1955.

Contents: 1. The Anatomy of Genital Prolapse. 2. The Aetiology of Genital Prolapse. 3. The Varieties of Genital Prolapse. 4. The Symptoms of Prolapse. 5. Non-Operative Menagement. 6. Fothergill Operation and the Colporrhaphies. 7. The Modified Vaginal Hysterectomy. 8. Occasional Repair Operation. 9. The Complications of Repair Operations. 10. Postoperative Infections. 11. The Disturbances of Micturition Associated with Genital Prolapse. 12. The Rectal Complications of Prolapse. 13. Retroversion of the Uterus. Index.

The literature of genital prolapse and its management is very extensive and shows a continued and progressive development of the methods available for the cure of the condition. Much of what has been written about prolapse in the past is still valid, and all modern methods are based on the work of many pioneers such as Fothergill, Mayo, Ward, Bonney and others.

With the publication of this excellent, though inadequately illustrated, review of genital prolapse and allied conditions, the author is to be congratulated on presenting a work based primarily on a personal experience gained from some 7,000 vaginal plastic operations.

In the chapter on the 'Aetiology of Prolapse' due consideration is given to the causative factors resulting from childbirth, and the rather interesting point is made that lacerations of the birth canal are of less importance than overdistension of the canal beyond the limits of tolerance. When discussing 'Non-operative Management of Prolapse' the value of re-educative exercises is discussed at some length, and in summing up the author states 'that it is of distinct value in recent puerperal patients and in some cases of stress incontinence associated with milder degrees of genital relaxation.' Mr. Malpas unfortunately makes no mention of the value of these pelvic-floor exercises as a prophylactic measure. The sections on the operative treatment of prolapse and allied conditions are concise and excellent, as is the chapter on the complications of repair operations.

This important book contains a vast amount of valuable information and represents a prodigious effort on the part of a world-famous gynaecologist.

A.M.M.

RADIOLOGY

Recent Advances in Radiology. By Thomas Lodge, M.B., Ch.B. (Sheff.), F.F.R., D.M.R. Third Edition. Pp. 358 + x, with 182 illustrations. 45s. London: J. & A. Churchill Ltd. 1955.

Contents: 1. Neuro-Radiology. 2. Injuries and Diseases of Bone. 3. Diseases of Joints. 4. The Alimentary Canal. 5. Diseases of the Large Bowel—The Biliary Tract—Pancreas. 6. The Cardio-Vascular System. 7. The Lungs. 8. The Lungs. 9. The Lungs. 10. The Genito-Urinary System.

In his preface the new author of this standard book stated that it has been written from the 'standpoint of the radiologist working in the general hospital' rather than the specialist radiologist. The author has achieved his self-set task admirably and the reviewer has no doubt that the book will have a far wider scope and will be of value to the practising physician and surgeon. In presenting a review of modern radiological diagnosis the book is worth while. The high standard laid down by the original author, Dr. Kerley, has been well maintained and selection of subjects has been extremely well balanced. The reproductions are good and it is refreshing to see that a slavish attempt to produce diapositives (negatives) at the expense of detail has been avoided. Indeed in deductive as opposed to empirical radiological diagnosis the positive images should help rather than detract.

The publishers, Messrs. J. & A. Churchill have maintained the excellent standard that has become associated with their publications and few radiologists cannot gain from a study of this text.

The Recent Advances in Radiology can be recommended to a far wider field of medical practitioners than the author has stated in his preface and students of radiology can gain much from a study of this book.

E.S.

DYNAMIC PSYCHIATRY

The Practice of Dynamic Psychiatry. By Juldes H. Masserman, M.D. Pp. 790 + xxx. £5 2s. 0d. Philadelphia & London: W.B. Saunders Company. 1955.

Contents: 1. The Clinical Investigation of Human Behaviour. 2. Dynamic Evaluation of Clinical Syndromes. 3. Case Studies and Communications. 4. Clinical Dynamics of Affect and Self. 5. The Practice of Dynamic Psychotherapy. Appendices.

Some years ago the author published a book entitled 'Principles of Dynamic Phychiatry' in which he 'attempted to correlate various physiologic and psychologic concepts of behaviour into a comprehensive system termed "Biodynamics". The present volume extends the application of biodynamics to clinical psychiatry and to the theory and practice of medicine and its specialties'. The field covered is very wide indeed, and the final section of the book even deals with the psychology of politics, economics, and social problems.

Part I deals with the rationale, objectives, and methods of the psychiatric interview. There is a particularly good description of the approach to a psychiatric patient, and the techniques of securing a psychiatric history, and a useful short account of the various psychological tests and their functions.

Part 2 presents a description of various syndromes of behaviour disorder, including the neuroses, character disturbances, hysterical syndromes and psychosomatic disorders, mental deficiency and the psychoses. These are dealt with from the standpoint of the mental forces at work rather than of classification.

Part 3 deals with hospital records and psychiatric reports to referring physicians and to non-medical personnel such as courts insurance companies, and employment boards, which are admirably adapted to their recipients.

Part 4 is predominantly theoretical.

Part 5 constitutes nearly half the book, and is a survey of both the theory and practice of psychotherapy from ancient to modern times. There is much that is erudite in this section, not the least interesting part being that dealing with ancient practices such as the administration of shock treatment in Grecian times by means of electric eels!

There is a section on the evolution and present concepts of psycho-analysis and a review of the theory and practice of the schools of Adler, Jung, Alexander, and others. These are followed by a well-rounded account of the aims, methods, strategy, and tactics of biodynamic psychotherapy. This is based on the psycho-analytic approach, with variations of technique, and the use of drugs, hypnosis, group-therapy, and changes of environment where considered advisable.

In a book of this scope there must of necessity be sections which are dealt with too briefly, but there is a large amount of interesting case material illustrating many points both of diagnosis and therapy, and a bibliography of many hundreds of titles.

This book is stimulating, interesting, and eminently readable.

W.S.

MEDICAL, PHYSIOLOGICAL AND RELIGIOUS HEALING

New Concepts of Healing. Medical, Physiological and Religious. By A. Graham Ikin, M.A., M.Sc. Pp. 186 12s. 6d. London: Hodder & Stoughton. 1955.

Contents: 1. Healing—Medical, Physical, Psychological and Spiritual. 2. The New Testament and Healing. 3. Psychotherapy and the Church's Ministry of Healing. 4. Psychotherapy and Spiritual Direction. 5. Co-operation Between the Medical Profession and the Churches. 6. Some Healers To-day. 7. Spiritual Healing and the New Physics. 8. Suffering and Service. Appendices.

This is a sincere attempt by Miss A. Graham Ikin, a psychologist, to prepare the way for what the Bishop of Lincoln in his introduction refers to as 'full cooperation between clergy, doctors and psychotherapists in the work of healing and wholeness'. The author is well prepared for her task by her training in psychology, her own experience of sickness and her previous post as organizing secretary and lecturer for the Archbishop of York's Committee of Doctors and Clergy and the National Council for Pastoral and Medical Cooperation.

Her approach to the problem is represented by the following quotation from her own preface: 'Our prides and prejudices, our fears and our resentments, our hates and our touchiness, are as potent a cause of disease as any microbe. Spiritual healing involves a casting out of negative and infantile emotions and a growth towards spiritual maturity.' It is likely that all sincere doctors find themselves on common ground with this statement.

The book is recommended for students in the theological colleges and medical schools. There is no doubt about the sincerity and careful consideration which the author has put into her subject and the book contains much useful information in appendices covering some of the well-known organizations for spiritual healing.

In the mid-twentieth century there is probably much more common ground for religion, psychology and medicine than is usually believed. Outstanding difficulties are, in the reviewer's opinion, largely a matter of semantics arising from the very different type of training approved in these 3 professions. This difficulty is well seen in the chapter on 'Some Healers To-day'. The phraseology here will not carry conviction to a medically trained mind although the case histories could probably quite well have been recounted by a medical man in such a way as to carry very much more conviction. On the other hand the chapter immediately preceding, on 'Co-operation between the Medical Profession and the Churches', and much else in the book is well stated for the consideration of any open-minded doctor. Because of the great importance of this subject it is now up to the medical profession to state its own case for cooperation in equally sympathetic terms.

J.F.B.

HYPNOTIC SUGGESTION

Hypnotic Suggestion. Its Role in Psychoneurotic and Psychosomatic Disorders. A Thesis by S. J. van Pelt, M.B., B.S. Pp. 95 with illustrations. 8s. 6d. Bristol: John Wright & Sons Ltd. 1955.

Contents: Part I. 1. Preliminary Introduction. 2. Short Historical Outline of Hypnotism. 3. The Nature of the Hypnotic State. 4. The Incidence of Susceptibility to Hypnosis. 5. The Phenomena of Hypnosis. 6. Methods of Inducing Hypnosis. *Part II.* 7. The Aetiology and Mechanism of the Psychoneuroses. 8. The Role of Hypnotic Suggestion in the Treatment of Psychoneurosis. 9. Illustrative Case Histories. *Part III.* 10. General Summary of Thesis.

Every general practitioner should read this clearly and simply written book. Dr. van Pelt, President of the British Society of Medical Hypnotists, exposes the simplicity of the techniques required to get to the root cause of many anxiety states, psychoneuroses and psychosomatic disorders. Contrary to the practice of many hypnotists he claims that *light* hypnosis is all that is necessary in most cases and that hereby in 95% of people can be achieved in a matter of weeks what may take years by psychoanalysis. He propounds his theories of the aetiology and mechanism of the psychoneuroses and the role of hypnotic suggestion therein. He states that hypnosis is a form of super-concentration of the mind, and that any of the recognized forms of psychoneurosis is really a state of self- or accidental hypnosis. Thus an idea of real emotional significance will act as, and have the power of, a hypnotic suggestion, and a psychoneurosis is the same in its structure as the behaviour and symptoms arising from a hypnotic or posthypnotic suggestion. In the treatment thereof he prescribes 'Relaxation, Realization and Re-education'. Dr. van Pelt would not be in his presidential position if this book were not convincingly suggestive of the importance and role of hypnosis in psychotherapy. The 12 illustrative case histories give the reader a clear picture of this role and it is interesting to observe that the number of sessions required in therapy vary from 4 to 12. His claims in treatment appear reasonable. It would have been difficult to accept a claim of success by one treatment in a psychoneurotic with a complicated network of emotional disturbances, as less experienced hypnotists aver. It reminds one of the surgeon who performs a herniorraphy and claims success because he never sees the patient again, little realizing that his associate around the corner may have repaired his failures. As a non-hypnotist, this reviewer would more readily accept many claims of success in hynosis when theories are re-inforced by facts and by controlled experiments.

R.S.

STAMMERING

Stammering: Its Cause and Cure. A Supplement to Stammer is is not Nerves. By H. V. Hemery, L.R.A.M. Pp. 17 + iv with 5 illustrations. London: The School for Functional Speech Disability. 1955.

Contents: 1. The Basis of Speech. 2. Contradiction of Previous Theories. 3. The Postulation of a Basic Physical Error. 4. The Development of the Error. 5. The Nature and Definition of Stammering. 6. The Effects of Stammering. 7. The Correction of Stammering.

Contents: 1. The Purpose of the Supplement. 2. Public Opinion. 3. Medical Views. 4. Some Stammer; Others copy. 5. Stammering is being Cured. 6. Apparent Setbacks. 7. The Development of Customary Speaking Skill. 8. The Development of Stammering Skill. 9. The Re-education of the Stammerer.

The author's continuous contact with his pupils may explain the degree of repetition throughout this book (and the supplement, which, in turn, is a modified repetition of sections of the book) which makes reading very difficult. In attempting to create a new science out of the art of teaching, Mr. Hemery has failed miserably. Despite his warning to readers not to confuse 'nerves' with nervousness he errs himself. He states that "stammering is a physiological bad habit which has its roots in basic error'. This 'basic error' is used throughout the book as some mysterious undefined etiological concept or even as an actual lesion, not to be confused with 'wrong motor inco-ordination'; yet he states elsewhere (and often) that the bad habit (of stammering) is due to wrong motor inco-ordination, and this 'bad habit' in effect, and in fact one discovers, turns out to be the 'basic error', e.g. on p. 38—'It cannot be emphasized too strongly that it is precisely the existence of the basic error and the lack of its replacement by a serviceable motor-habit that is the bane of the stammerer'. In the eyes of the reviewer a habit (good or bad) is based upon a reflex which depends upon the integrity of the nervous system. In fact the author himself proves that 'stammer *is* nerves'. The practical aspects of the re-education of the stammerer expounded in the Hemery Method of Speech

Re-education for Stammerers are not disputed by the reviewer because they embody the generally accepted principles of correction of speech defects. It seems that this book has been written merely as a vehicle to expound on the Hemery Method. The book could have been reasonably and readably condensed into about one-third of its present size and most of the diagrams and figures, which are un-anatomical, un-physiological and unscientific, could have been dispensed with. These two books, written for stammerers and their teachers, merely confuse the confounded.

R.S.

CORRESPONDENCE : BRIEWERUBRIEK

THE SPECIALIST REGISTER

To the Editor: The letter on this subject above the nom-de-plume *Sen. G.P.* in the *Journal* of 14 April contains a lengthy extract from the *Family Doctor*. Despite the source one cannot fully agree with the sentiments expressed.

The concluding sentence of the quotation—'Choosing your own specialist is a bit like playing Russian roulette with one round in a six-chambered revolver. It might work out fine—or it might not'—sounds like the Pythian oracle! It surely must apply to the choice of the G.P. as well!

Because the *Family Doctor* is a publication for the layman it advocates, perhaps wisely, that the recommendation of a 'specialist' should come from the G.P. But nepotism in this direction is not unknown! Besides, so many people have no regular G.P., for it is the era of specialization we are living in; or they are visitors to the town, asking the advice of a friend, chemist or hotel porter. Or it may be that the patient, while preferring to obtain an independent opinion does not want to offend his G.P. While in specialist practice in Johannesburg I even had the experience on several occasions of being given a false name and address.

But is it not the patient's *right* to choose his medical adviser, which means '*free choice*'? 'You pays your money and you takes your choice'!

I fully endorse the concluding words of *Sen. G.P.*: 'Free choice of doctor is a motto designed to emphasize our opposition to closed panels; it cannot legitimately be used in a question of another kind altogether—the patient/doctor/specialist relationship'.

The reasoning by the *Family Doctor*, as quoted, is unhappily illogical. A neuro-surgeon, consulted for headache, will take the blood pressure (he may even test the pressure of the cerebrospinal fluid) and also test the urine. Is not the specialist on the register in South Africa required to be first a G.P. before he can be registered?

By now myself a 'senior' and no longer on the specialist register, but again a G.P., I will urge that it is only *success* that counts with our patient. This can never change. If you can help him you are a 'good doctor', and if you cannot help him in the way he expects you are a 'bad specialist'.

Some of the arguments on the subject of 'Specialist Register' in this column were illogical. No wonder that the honest and logical article by Dr. James Black carried the day! It was most reassuring to learn that the older, experienced, level-headed members are still in the majority on the Council. Let us hope that this happy state of affairs will persist; it naturally rests entirely with ourselves, casting our vote for the right representatives available. Even if the election is still far off we must already now make up our minds in this direction and persuade suitable colleagues to accept nomination for these arduous duties.

In conclusion: We must never forget that our ancient motto is still *salus aegroti suprema lex* even if it will mean no entry into the ledger!

Nathan Finn

27/30 C.N.A. Building
East London, C.P.
20 April 1956

DIPLOMA IN PSYCHOLOGICAL MEDICINE

To the Editor: I have been instructed by the Specialists Committee of my Council to request you to be good enough to advise the profession, through the medium of your esteemed *Journal*, of the fact that the Diploma in Psychological Medicine will be accepted as a higher qualification for the speciality Neurology only until 1 January 1958, and that thereafter recognition of this Diploma as a higher qualification for the speciality Neurology will be discontinued.

The Committee will also be pleased if you will make it known that a revised and published list of qualifications acceptable as higher qualifications in terms of the Council's rules for the registration of specialities, is now available; copies thereof may be obtained from the offices of the Council.

W. H. Barnard
Asst. Registrar

The South African Medical and Dental Council
P.O. Box 205
Pretoria
23 April 1956

COPIES OF THE B.M.J.

To the Editor: I have copies of the B.M.J. (approximately 3 years) which I am prepared to give away. Anyone who would care for these and is prepared to pay railage, should communicate with me at this address before 7 May, at which date I shall be leaving here.

R. Fry

Irrigation Department
Bothaville, O.F.S.
4 April 1956.

REPEAT LAPAROTOMY FOR AN ECTOPIC PREGNANCY

To the Editor: It may be of interest to some of your readers to learn about the follow-up of the patient in the article[1] published under this title in August 1954. To recapitulate, a right salpingectomy was performed on 20 September 1953 for ectopic pregnancy. Recurrence of lower abdominal pain and signs and symptoms of internal haemorrhage necessitated a repeat laparotomy (in another town) in December 1953, when right cornual rupture was found. Cornual excision was performed after first placing mattress sutures *in situ* and oversewing the area with broad-ligament peritoneum.[2] It was concluded that a haematosalpinx only was removed at the first operation.

The patient subsequently became pregnant with an intrauterine gestation. She was admitted to the Maternity Block of the Provincial Hospital, Port Elizabeth, with pre-eclampsia in late pregnancy. This was kept under control by rest and sedation during the last 4 weeks of her pregnancy. Because of the cornual excision, watch was kept for uterine rupture, She had a spontaneous delivery of a normal female infant in April 1955, with a normal third stage. No untoward occurrence has been reported to date.

A satisfying feature of this case is the strength of the cornual scar in pregnancy so soon after the lesion occurred. Recently, at the Livingstone Hospital, Port Elizabeth, by coincidence, 4 ectopic pregnancies occurred in one month, where the cornual area had to be excised. In all, the method of pre-excision mattress sutures was used with re-peritonizing of the area by the broad-ligament peritoneum. Further, I now instil a few centimetres of normal saline down the unaffected tube at the conclusion of the operation for ectopic pregnancy. In this way, I hope to lessen the chance of a future ectopic. It would be interesting to hear if any other practitioners have experience of this procedure. Finally, while one is well aware of the common causes of ectopic pregnancy, it is presumed that the toxaemia in this patient was a coincidental feature, and not linked with a common etiological feature of endocrine imbalance that was responsible for the previous ectopic pregnancy.

James Miller

4, Western Road
Port Elizabeth
25 April 1956

1. Miller, J. (1954): S. Afr. Med. **28**, 750.
2. *Idem.* (1953): *Ibid.*, **27**, 246.